UPPER GASTROINTESTINAL BLEED- CAUSES, ENDOSCOPIC PROFILE AND USEFULNESS OF ROCKALL SCORE

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CERTIFICATE

Certified that this dissertation titled **"UPPER GASTROINTESTINAL BLEED- CAUSES, ENDOSCOPIC PROFILE AND USEFULNESS OF ROCKALL SCORE"** is the bonafide record work done by **Dr. REMA KRISHNAKUMAR**, during the period 2005-08, under my guidance and supervision and is submitted in partial fulfillment of the requirement for the DM (Branch – IV) Medical Gastroenterology, of The Tamil Nadu Dr. M.G.R. Medical University, August 2008 examination.

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CONTENTS

Criteria	Abbreviation	Expansion
Presentation	H/M	Hematemesis/ Melena
Severity	MN/MD/MS	Minor/Moderate/Severe
Associated factors	A/S/N/C/B	Alcohol/Smoking/NSAID/Corrosive/ Bleed in past
Comorbidity	C/R/L/O	Cardiac/Renal/Liver/ Others
Timing of endoscopy	E/L	Early/ Late
Endoscopic findings	O/G/D/MW/DU/GU/ MS/A/V/PHTG/ PHTD	Oesophagitis/Gastritis/Duodenitis/ Mallory –Weiss tear/Duodenal ulcer/ Gastric ulcer/ Malignancy/ Angiodysplasia/ Varices/Portal hypertensive gastropathy/ Portal hypertensive duodenopathy
Risk group	A/B/C	Low / Moderate/High
Transfusion	B	Blood transfusion
Endotherapy	A/EST/EVL	Adrenaline/Endoscopic Sclerotherapy/ Endoscopic variceal ligation
Rebleed	Y	Yes
Mortality	D	Death

CHAPTER 1

INTRODUCTION

Upper gastrointestinal bleeding (UGIB) is a common medical emergency associated with significant morbidity and mortality and forms a bulk of admissions to medical centres and may encompass many different scenarios. A broad spectrum of lesions may be responsible for bleeding from the upper gastrointestinal tract; the bleed being massive or trivial and either clinically apparent or obscure.

Gastrointestinal (GI) bleeding is an extremely common clinical problem resulting in more than 3,00,000 hospitalizations annually in the United States. The overall incidence of upper GI bleeding is approximately 125 hospitalizations for every 1,00,000 people, with a male to female ratio of 2:1. [1] Bleeding from upper GI tract is five times as common as lower GI bleed.

Over the past 45 years, the mortality from upper GI bleeding has remained stable at approximately 10%.[2] Mortality from acute GI bleeding is much greater than that for chronic bleeding. Therefore, it is important to understand the pathogenesis of acute GI bleeding, with an emphasis on early detection, prevention and intervention, in order to minimize morbidity and mortality.

The clinical presentation reflects the site, etiology and rate of bleeding and may manifest in one or more ways. Hematemesis, melena and hematochezia are the most common manifestations. The bleeding may be obscure in about 5% of cases and at times may manifest as an occult bleed.

Peptic ulcers are the most common causes of upper GI bleeding and are followed by variceal bleeding, gastric and duodenal erosive disease and

Mallory-Weiss tears in prevalence. The associated factors include H. pylori infection, NSAID intake and alcohol abuse.

An initial hemodynamic assessment helps to plan resuscitation, forms the basis of further management and also predicts the prognosis of the patient. Analysis of clinical and endoscopic factors permits accurate risk assessment, rational treatment planning and improved outcome.

Early upper gastrointestinal endoscopy, defined as within 24 hours of hospital presentation or admission is the cornerstone of management of UGIB. Early endoscopy helps in diagnosis, treatment and risk stratification. Therapeutic endoscopy is considered a safe and effective form of treatment today. [3]

A number of scoring systems have been designed to ascertain risk factors for poor outcome and to improve patient management and promote cost-effective use of hospital resources in patients with UGIB. Rockall et al. developed a risk-scoring system involving clinical and endoscopic criteria to predict the risk of rebleeding and mortality in patients with UGIB. It is based on age, presence of shock, co-morbidity, diagnosis and endoscopic stigmata of recent hemorrhage. Multiple studies have validated the Rockall score's ability to identify and risk-stratify patients with UGIB. The Rockall system has been shown to represent an accurate and valid predictor of rebleeding and death. This has the potential to result in a more appropriate management of subjects' conditions based on their assessed risk of complications following the initial UGI bleed.

CHAPTER 2

AIM OF THE STUDY

(i) To study predisposing factors, clinical profile and endoscopic findings of patients presenting with Upper Gastrointestinal Bleed (UGIB) to the endoscopic unit of our institution

(ii) To apply Rockall score in the assessment of upper gastrointestinal bleed

CHAPTER 3

REVIEW OF LITERATURE

Epidemiology

UGIB is a common reason for emergency admission to hospitals. A recent large prospective study from the United Kingdom reported an overall incidence of 103 per 1,00,000 adults per year with an overall mortality of 14%, but only 0.6% for those below 60 years of age without co-morbidity. Acute upper gastrointestinal haemorrhage accounts for about 2500 hospital admissions each year in the United Kingdom. The annual incidence varies from 47 to 116 (approximately 100) per 1,00,000 population and is higher in socioeconomically deprived areas.[4] The incidence is approximately 72 per 1,00,000 population in Malaysia.[5,6] A retrospective study from USA also showed a similar incidence of 102 per 1,00,000 adults.[7]

The incidence increases markedly with age. Consequently, many patients presenting with UGIB have an active comorbid condition, a consistent risk factor for increased mortality. Rockall and Logan[8] et al. and Yavorski [9] et al. noted a mean age of 66 years and 52 years respectively, in their series. Longstreth and colleagues[10], in their series, noted that 47% of their patients were above 60 years of age.

The incidence of UGIB is twice as high in men as in women. Barkun et al.[11] noted that 62% were males. Rockall et al. observed a male preponderance of 57%. Longstreth et al. also have noted a male predilection of 67.9%.

Hospital mortality has not improved over the past 50 years and remains at about 10%.This may in part be due to the fact that older patients, who have advanced cardiovascular, respiratory, or cerebrovascular disease that puts them at increased risk of death, now comprise a much higher proportion of cases. Much of the morbidity and mortality of UGIB occurs in patients with recurrent bleeding or significant co-morbid illnesses.[12] Figures available from a small prospective study from Singapore have showed an overall mortality of 10%. [7]

Modes of presentation

Hematemesis, melena and hematochezia are the most common manifestations.

Hematemesis is defined as the vomiting of blood and is caused by upper gastrointestinal bleed from the oesophagus, stomach or small bowel. The blood may be bright red or may take on the appearance of coffee–grounds. Patients with coffee ground emesis are not usually bleeding actively but have had a recent or even remote bleed.

Melena is defined as passage of black, tarry and foul smelling stools, the tarry character being caused by the degradation of blood in the more proximal colon and is typical of bleeding from upper GI tract. It is caused by delivery of at least 50 ml of blood into the upper gastrointestinal tract and indicates that blood has been in the gastrointestinal tract for extended periods of time.

Hematochezia is the passage of bright red blood per rectum that may or may not be mixed with stools.

Barkun et al. [11] noted hematemesis in 58% of patients and melena in 69% and Longstreth et al. [10] observed that 33% of their patients had hematemesis and 81% had melena. Hematochezia was noted by Barkun et al. in 15% and Laine et al. in 5% of their cases.[13]

Obscure gasrointestinal bleed, is generally accepted to be GI bleeding that persists or recurs without an etiology after standard endoscopic examination, and occurs in about 5% of patients with GI bleed.

Occult GI bleeding is taken to mean bleeding that is truly unknown to the patient and may manifest with symptoms of blood loss like dizziness, dyspnoea, angina or even shock without any objective signs of bleeding. Features of iron deficiency anemia or fecal occult blood positivity may be present.

ETIOLOGY OF UPPER GASTROINTESTINAL BLEEDING [14]

NON-VARICEAL CAUSES

Oesophagus

- Mallory-Weiss tear
- Severe oesophagitis
- Oesophageal ulcer
- Cameron ulcer within hiatus hernia
- Oesophageal neoplasm
- Infections- bacterial, viral and fungal infections

Stomach

- Gastric ulcer
- Gastric erosions

- Gastric malignancy
- Others- gastric polyps, Dieulafoy lesion, angiodysplasia

Duodenum

- Duodenal ulcer
- Duodenal erosions
- Vascular malformations
- Aorto-enteric fistula
- Polyps (including Peutz- Jeghers syndrome and other polyposis syndromes)
- Carcinoma of ampulla, carcinoma of pancreas, haemobilia

Small bowel

- Stomal ulcer
- Diverticulae (including Meckel's diverticulum)
- Vascular malformation, tumours

Bleeding caused by portal hypertension

- Varices
- Nonvariceal mucosal lesions- portal hypertensive gastropathy, gastric antral vascular ectasia

Peptic ulcer disease

Peptic ulcer disease accounts for 50% to 70% of cases of acute nonvariceal upper GI bleeding .[15, 16]

Ulcer bleeding starts when the ulcer base erodes into a blood vessel, and the severity of the bleed is dependent on the size of the vessel affected.

Simple oozing is caused by damage to small submucosal vessels less than 0.1 mm in diameter and more severe arterial bleeding indicates that a large vessel between 0.1 and 2 mm in diameter in the base of the ulcer has been eroded by the inflammatory process. Large ulcers arising from the posterior part of the duodenal cap can erode the gastroduodenal artery and provoke brisk bleeding.

Spontaneous hemostasis occurs when a sentinel clot plugs the “side hole” in the vessel. The clot may then enlarge, remain attached for some time as it organizes, and eventually sloughs off, leaving the underlying vessel covered with a flat pigmented spot that fades to leave a clean ulcer base This process takes less than 72 hours, and rebleeding occurs if the clot undergoes lysis or falls off prematurely.[17]

Even though ulcer bleeding stops spontaneously in at least 80% of patients, the overall mortality is unchanged over the last 30 years, ranging from 6 to 7% in the United States [13] and averaging 14% in the United Kingdom.[18] Without specific hemostatic intervention, peptic ulcer bleeding continues or recurs in approximately 20% of patients.

Erosions

Acute erosive gastritis can cause persistent haemorrhage as a result of diffuse loss of mucosal epithelium and is often associated with the use of non-steroidal anti inflammatory drugs, steroids and intake of alcohol. Haemorrhagic gastritis occurring as a result of impaired mucosal blood flow is often caused by stressful stimuli including shock, hepatic failure and head injury.

Oesophagitis usually only causes minor acute bleeding. Occasionally a significant vessel may be involved with consequent massive arterial hemorrhage.

Mallory-Weiss Tear

Mallory-Weiss lesions are tears occurring at or near the esophagogastric junction, secondary to mechanical stress most commonly induced by vomiting and increased intra abdominal pressures during retching.

Mallory-Weiss lesions account for 4–14% of all cases of acute upper GI bleeding in patients who undergo endoscopy.[19] Most series report a male predominance of 60–80%.[20] with mean age typically in the fourth to sixth decades. Recent alcohol ingestion has been reported in 21–80% of cases. Any condition causing vomiting could produce a tear, including coughing and pregnancy.

The diagnosis of Mallory-Weiss lesions is best made endoscopically with close inspection of the gastro-oesophageal junction. The lesion is longitudinal, most commonly along the cardia, extending proximally to include the distal esophagus. Occasionally, repeated vomiting may result in a full thickness tear (Boerhaave's syndrome) which is associated with sudden onset of severe pain in the upper abdomen or chest.

The bleeding associated with Mallory-Weiss lesions is usually self limited, with spontaneous cessation of bleeding reported in 90% of cases. [20] Protracted bleeding can rarely occur.

Malignancy

Carcinoma and lymphoma of the stomach commonly bleed at an advanced ulcerated stage, and occasionally present with acute hemorrhage.

RISK FACTORS

Gastric Acid

The evidence for a role of gastric acid in peptic ulceration includes the hypersecretory disorder Zollinger-Ellison syndrome, in which patients develop ulcers with high frequency. [21]The ability of antacid therapy alone to heal upper gastroduodenal tract ulceration also supports the role of acid. Acid reduction by proton pump inhibitors in patients with active or recent bleeding from upper gastrointestinal ulcerative lesions reduces the risk of bleeding and rebleeding.[22]

Aspirin and other Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

Approximately 15-30% of patients exposed to NSAIDs develop gastroduodenal ulcers. The mechanism of injury and ulceration is complex but appears to involve reduced production of cytoprotective prostaglandins. Further, the risk of bleeding is increased in part because of platelet dysfunction.

The following points appear to be of note:

(1) The risk for gastric ulceration is greater than that for duodenal ulceration, although both are increased

(2) The risk of bleeding varies with the individual NSAID

(3) The risk of bleeding is dose dependent

(4) Multiple cofactors contribute to NSAID risk

- Age greater than 75 years
- History of heart disease
- History of peptic ulcer
- History of previous gastrointestinal bleeding

In addition, H. pylori may be a risk factor for ulcers. Corticosteroids, bisphosphonate (alendronate), and ethanol appear to potentiate the ulcerogenic effect of NSAIDs. Any dose of aspirin has the potential to cause gastrointestinal bleeding, the enteric-coated form carrying the same risk as plain aspirin .[24]

Helicobacter pylori

H. pylori(HP) is a gram negative bacteria whose motility and adherence mechanisms allow it to colonize the stomach despite ongoing gastric motility. The most dominant feature of HP is its ability to tolerate the stomach environment with its acidic pH, constant emptying and rapidly exchanging epithelial layer. Prevalence rates are higher in developing countries, and in these areas, infection is much more common in the young.

Role of H. pylori in ulcer bleeding is controversial. Studies have suggested that H. pylori infection increases the likelihood of hemorrhage (relative risk, approximately 1.5).[25]In contrast, one study revealed a decreased incidence of H. pylori infection in patients presenting with actively bleeding ulcers.[26]

The role of H. pylori infection in causing hemorrhage of ulcers in those using NSAIDs is also controversial. On one hand, NSAID users infected with H. pylori had a nearly twofold risk of ulcer bleeding compared with uninfected NSAID users. In contrast, other studies have suggested that H. pylori has little adverse effect or may even protect against NSAID–associated gastroduodenal lesions and promote ulcer healing. The eradication of H.pylori substantially reduces the risk of ulcers for patients who are about to start long-term NSAID therapy. [27]

Techniques to diagnose HP infection, such as serologic testing and histologic analysis, require several days for confirmation and may not be useful in the setting of acute bleeding. Only 2 tests are available to rapidly assess HP infection, the CLO (rapid urease) test and carbon 14 urea breath analysis. The breath analysis is not uniformly available in many institutions. Further, data suggest that in those undergoing endoscopy for active bleeding, the CLO test lacks sensitivity with a substantial false-negative rate. Lee et al.[23] analyzed the diagnosis of HP infection in 55 patients with bleeding duodenal ulcers and compared results with 69 patients with uncomplicated ulcers. A variety of diagnostic methods to assess HP infection were used including the CLO test, serologic analysis, and microbiologic and histologic evaluation. The false negative rate with the CLO test was significantly higher in those with bleeding ulcers vs those without (18.2% vs 1.4%; $P<.05$). They further noted that those with bleeding ulcers had HP infection rates significantly lower than those with uncomplicated disease (72.7% vs 92.8%; $P<.05$). These data would suggest that in the absence of breath analysis, a rapid and reliable method to diagnosis HP infection in those with bleeding ulcers is lacking.

Ethanol

Ethanol is well known to induce gastric mucosal injury and thus may cause or potentiate ulcer bleeding. Deleterious effects of NSAIDs are further increased among drinkers. Patients who ingest ethanol chronically may have alcohol-induced liver disease and secondary portal hypertension, which is an important risk factor for non ulcer upper gastrointestinal hemorrhage.

Anticoagulation Therapy

Anticoagulation increases the risk of bleeding from ulcer disease. The relative risk of hospitalization for bleeding ulcer in anticoagulated patients is about 3, and anticoagulants further increase the risk of bleeding in those taking NSAIDs. [28]

Longstreth et al.[10] noted history of NSAID use in 53% and alcohol use in 3% of patients, in their series.

BLEEDING FROM PORTAL HYPERTENSION

Portal hypertension may lead to bleeding from several different lesions, including esophageal varices, gastric varices, ectopic varices, portal hypertensive gastropathy and portal hypertensive duodenopathy.

VARICES

Oesophageal varices

They appear as serpentine venous channels that course through several levels from the lamina propria to the deep submucosa of the oesophagus, achieve their greatest prominence, as a rule, 2 to 3 cm above the gastro-oesophageal junction, and in time may extend cephalad to the mid-oesophagus(usually upto 24 cm from incisor teeth). Rupture of oesophageal varices is a common cause of life threatening hemorrhage.

Gastric varices

The next most common site for the formation of clinically significant varices is the stomach, either in obvious continuity with oesophageal varices, that is, true gastro-oesophageal varices, or as free-standing gastric varices. Various classifications for the different locations of gastric varices have been proposed. According to Sarin et al [30], gastro-oesophageal varices type I (GOV

I)are those that appear as an inferior extension of oesophageal varices; type II are isolated gastric varices(IGV) in the fundus or body and antrum of the stomach without esophageal varices.[29,30] GOV are further subdivided into GOV1(extending along the lesser curvature) or GOV2 (extending along the greater curvature towards fundus of stomach) Similarly, IGV have been divided into IGV1(located in gastric fundus) and IGV2(located in antrum, body and pylorus). GOV1 is the most common variant, accounting for 74%, followed by GOV2 (16%), IGV 1(8%) and IGV2 (2%).

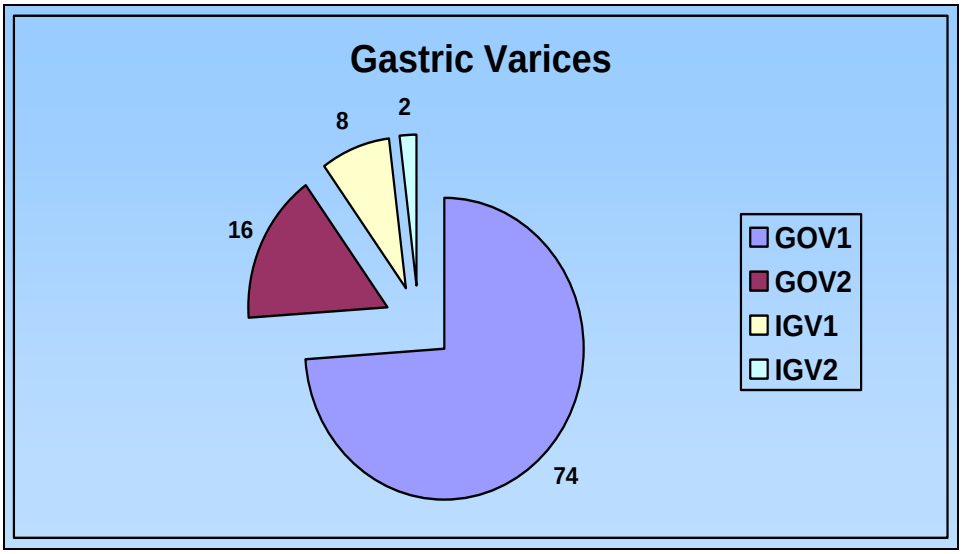


Fig 1.Sarin's classification of gastric varices

The appearances of gastric varices vary from a relatively subtle, bluish discoloration of otherwise normal appearing gastric mucosal folds to more obvious, classic cerebriform clusters of veins. The largest study to date found gastric varices in 20% of patients who had portal hypertension from a variety of causes.[30] Factors that promote the formation of gastric varices are poorly understood. Gastric fundal varices in the absence of oesophageal varices should raise the suspicion of splenic vein thrombosis, although in most cases

fundal varices are simply a manifestation of portal hypertension caused by cirrhosis.

Nonvariceal mucosal Lesions

Portal hypertension is associated with a widespread abnormality in gastrointestinal mucosal microcirculatory integrity. Portal hypertensive gastropathy is a common finding in patients who have portal hypertension.[31] Here, there is evidence for increased gastric mucosal blood flow .[32] Although previous sclerotherapy has been noted by some authors to increase the risk of gastropathy [31], there is little agreement on this point. [33]

Portal hypertensive gastropathy is often graded by endoscopic appearance from mild to severe, with mild gastropathy characterized by a mosaic or "snakeskin" pattern of erythema and severe gastropathy by a variety of morphologic characteristics including bright red punctate erythema, diffuse hemorrhagic lesions, and black or brown spots indicating submucosal hemorrhages. Portal hypertensive gastropathy accounts for 8% to 20% of acute bleeding in patients who have portal hypertension and also has been identified as an important source of chronic blood loss. [33]

Gastric antral vascular ectasia (GAVE), or "watermelon stomach," may occur with increased frequency in cirrhosis and portal hypertension (40%). The lesion may be confused with portal hypertensive gastropathy but shows little relation to portal hypertension and is not ameliorated by portal decompressive procedures.[34]

Large national surveys have reported that about 10% of patients with UGIB bleed from varices[35] whereas in inner city hospital populations, approximately one third of patients bleed from varices.[36]

Rockall and Logan [8] studied 2332 cases of UGIB, taking into consideration both variceal and non variceal etiology of bleed and the various causes included peptic ulcer-842(36.1%), erosive gastritis-240(10.3%), oesophagitis-241(10.3%), Mallory-Weiss tear-119(5.1%) ,malignancy- 93(4%) and varices -108(4.6%)

Barkun et al. [11] noted in their study based on RUGBE database, that 56% had peptic ulcers disease as the primary etiology for UGI bleeding, followed by oesophagitis (8.4%), Mallory- Weiss tears(4.4%) and Dieulafoy lesions(2.5%)

INITIAL PATIENT ASSESSMENT

The first step in treating all patients with gastrointestinal bleeding is to assess the severity of bleeding. Therefore, hemodynamics is the initial focal point, helping to focus resuscitation efforts and predicting prognosis.

Resuscitation

Airway

A drowsy or comatose patient is at high risk of aspiration if vomiting or hematemesis continues. The patient is kept flat on his/her side. A cuffed endotracheal tube may be inserted to protect the airway if needed.

Mental status may be impaired due to

1. Cerebral hypoperfusion due to severe acute blood loss
2. Encephalopathy due to concomitant chronic liver disease or renal failure
3. Alcohol or drug intoxication/overdose

When a patient presents with gastrointestinal bleeding, risk assessment and resuscitation should proceed simultaneously. The first step in management

should be to assess the severity of bleeding. Upper GI bleed can be categorized into minor, moderate or major, depending on hemodynamic assessment. [37]

Table 1. Severity of bleed

Vital signs	Loss of intravascular volume (%)	Bleed type
Shock (resting hypotension)	20-25	Massive
Postural (orthostatic tachycardia or hypotension)	10-20	Moderate
Normal	<10	Minor

This categorization forms the basis of further management, focuses rescuscitation efforts, provides important prognostic information and helps triage appropriate intervention.

In patients with hemodynamic instability, two large bore IV catheters should be placed, normal saline or Ringer lactate solution infused along with packed cell transfusions to raise hematocrit to 30% in elderly and 27-28% in those with portal hypertension. Supplemental oxygen should be given along with monitoring of central venous pressure, vital signs and urine output.

History and examination

The history helps the clinician assess the severity of bleeding and make a preliminary assessment of the site and cause of bleed.

Physical examination may reveal the presence of cutaneous signs (spider angiomata, Dupuytren's contracture) or other evidence of liver disease (splenomegaly, ascites, caput medusae), that suggest the possibility of portal

hypertension. Acanthosis nigricans may reflect underlying gastric cancer; cutaneous telangiectasias of skin and/or mucous membranes and lips may point to hereditary hemorrhagic telangiectasias (Osler-Weber-Rendu syndrome); pigmented lip lesions are seen with Peutz-Jeghers syndrome; cutaneous tumors suggest neurofibromatosis; purpura is consistent with vascular disease (Henloch-Schönlein purpura or polyarteritis nodosa).

Abdominal tenderness (peptic ulcer, pancreatitis, ischemia), abdominal masses, lymphadenopathy (malignancy), and splenomegaly (cirrhosis, splenic vein thrombosis) are all important to detect, in a case of UGIB.

Bedside examination of the character of the stool provides critical information not only about the site of bleeding, but also about the acuity of bleeding. Patients with brown stools are unlikely to have aggressive bleeding. In contrast, patients who are actively passing stools containing red blood, maroon-colored blood, or melena even in the absence of a positive nasogastric lavage; are likely to have active bleeding. Those with a history of coffee ground emesis only and normal-appearing stools, often positive for occult blood, have usually had a trivial bleed.

The blood urea nitrogen (BUN) level may be mildly elevated in patients with upper GI bleeding. The elevation is typically out of proportion to elevation in the serum creatinine level [38] due to breakdown of blood proteins to urea by intestinal bacteria and its absorption, as well as from a mild reduction in glomerular filtration rate.

The nasogastric lavage has been used extensively to help differentiate upper from lower gastrointestinal bleeding. [27] A bloody aspirate confirms the upper gastrointestinal tract as the source of bleeding, since the false-positive

rate is essentially nil and is related to nasogastric trauma.The correlation between the acuity of bleeding and the physician's assessment of bleeding is weak, with a 79% sensitivity and 55% specificity for active bleeding. It is negative in up to 25% of patients with upper gastrointestinal bleeding.

RISK STRATIFICATION

At the initial assessment it is important to define the factors that have prognostic importance.

Risk factors for death after hospital admission for acute upper gastrointestinal bleeding [18]

1. Advanced age
2. Shock on admission (pulse rate >100 beats/min; systolic blood pressure < 100 mm Hg)
3. Comorbidity (particularly hepatic or renal failure and disseminated malignancy)
4. Diagnosis (worst prognosis for advanced upper gastrointestinal malignancy)
5. Endoscopic findings (active, spurting haemorrhage from peptic ulcer; non-bleeding visible vessel)
6. Rebleeding (increases mortality about 10 fold)

The main factors predicting death include increasing age, comorbidity and endoscopic findings. Mortality is low in patients below 40 years of age but increases steeply thereafter. Patients with severe comorbidity, particularly renal failure, liver failure and disseminated malignancy have a poor prognosis. Death in these patients is more often due to disease progression rather than due to the upper gastrointestinal bleeding. Patients who developed UGIB

after hospitalisation for other serious illnesses have a much worse prognosis than those who are admitted because of bleeding, with a mortality of about 30%.

Endoscopy for risk assessment

Endoscopy is the most accurate method available for identifying the source of bleeding and providing therapy in UGIB. A large double or single channel therapeutic endoscope should be used in all suspected cases of upper GI bleed.Positioning the patient with bleeding point in the most superior position can help clear the endoscopic field by allowing blood to flow away from the point of bleeding. Reverse Trendelenberg positioning and rolling patient from left lateral decubitus position to the back can also be used to move clots away from dependent areas of the stomach.

Early upper gastrointestinal endoscopy , defined as within 24 hours of hospital presentation or admission; is the cornerstone of management of UGIB. Early endoscopy has 3 major roles viz. diagnosis, treatment and risk stratification. It has been shown to reduce resource use, decrease transfusion requirements and shorten hospital stay. Chak et al. observed that early endoscopy was done in 82% of their patients.[39] Rockall et al. noted that 1108(50.1%) of their patients underwent early endoscopy. Longstreth et al. suggested that early endoscopy is an important factor in shortening duration of hospital stay, identifying patients for outpatient care and reducing costs in upper GI bleed. Lee et al. stated that early endoscopy was most accurate in determining the source of bleeding and reduced transfusion requirements and length of hospital stay.

Endoscopic findings of active, spurting haemorrhage and a non-bleeding visible vessel within an ulcer are associated with a definite risk of rebleeding. The absence of these stigmata, varices or upper gastrointestinal cancer indicates a low risk of rebleeding. Ulcers located in postero-inferior portion of duodenal bulb and high on lesser curvature of the stomach, have a poor prognosis.

Forrest Classification for Bleeding Peptic Ulcer [40] has been formulated, which helps to plan endoscopic treatment and assess risk of rebleed.

The classification is as follows

IA: Spurting Bleeding

IB: Non spurting active bleeding

IIA: visible vessel (no active bleeding)

IIB: Non bleeding ulcer with overlying clot (no visible vessel)

IIC: Ulcer with pigmented spot

III: Clean ulcer base (no clot, no vessel)

Laine et al.[13] noted, in their series that Forrest class I ulcers constituted 18%, Forrest IIA and B ulcers were found to contribute to 17% each, IIC ulcers constituted 20% and Forrest class 3 ulcers, constituted a majority (42%).

Risk of rebleeding and mortality

Peptic Ulcer Bleed[13]

Table 2.Forrest class and risk of bleeding

Endoscopic finding	Risk of Rebleeding (%)	Mortality (%)
Active bleeding	55	11
Visible Vessel	43	11
Adherent Clot	22	7
Flat Spot	10	3
Clean Base	5	2

Variceal bleed

The stigmata of recent hemorrhage(SRH) include red colour signs which include red “wale” markings, which are longitudinal whip-like marks on the varix; cherry-red spots, which usually are 2 to 3 mm or less in diameter; hematocystic spots, which are blood-filled blisters 4 mm or greater in diameter; and diffuse redness Patients with large oesophageal varices, Child-Turcotte-Pugh (CTP) class C cirrhosis, and red colour signs on varices have the highest risk of variceal bleeding within 1 year.

Recently, a number of studies have indicated that systematic assessment of clinical and endoscopic risk factors (endoscopic triage) may obviate hospitalisation in some patients and may help in determining the appropriate length of stay in others. [10, 41, 42] Those determined to be at low-risk based on clinical and endoscopic criteria were discharged on the day of presentation and received out-patient care. [43, 44] The aforementioned findings

have led to the development of practice guidelines and clinical care pathways for UGIB. [45, 46]

Use of Risk Stratification Scoring Systems

Although endoscopic findings can identify individuals at a high risk of rebleeding, overall mortality is often reflective of other factors such as age and co-morbid conditions. A number of scoring systems have been designed to ascertain risk factors for poor outcome and to improve patient management and promote cost-effective use of hospital resources in patients with UGIB.

Several clinical scoring systems e.g. Rockall score, Baylor bleeding score, the Cedar-Sinai Medical Centre Predictive Index and the Blatchford score, have been developed to direct appropriate patient management and enable cost effective use of resources. These systems weigh a combination of clinical, laboratory and endoscopic variables to produce a score that predicts the risk of mortality, recurrent haemorrhage, need for clinical intervention or suitability for early discharge.

Risk stratification using non endoscopic parameters has the advantage that it can be performed readily on initial presentation in the emergency department and appropriate initial risk assessment is still possible, even if early endoscopy, which requires skilled staff and resources, is not always available. Inclusion of endoscopic stigmata of recent hemorrhage (SRH) that relate to increased risk of re-bleeding and death into scoring systems increases the sensitivity for predicting patients at high or low risk compared to non-endoscopic assessments.[40, 42, 47] High risk lesions such as actively bleeding ulcers, non-bleeding visible vessels (NBVV) and adherent clots require effective aggressive intervention to reduce re-bleeding which is

associated with a 5-16 fold increase in mortality.[13,18] The rebleeding rate of ulcers with a clean base is low and endoscopic intervention is usually not recommended[48,49] In fact, early endoscopy-based triage may permit safe and early discharge of “low risk” patients with no increased rate of re-bleeding or mortality.[50]

Rockall et al [18] conducted a prospective, multicentre, population based study using standardized questionnaires in two phases one year apart. A total of 4185 cases of acute upper gastrointestinal hemorrhage over the age of 16, identified over a four month period in 1993 and 1625 cases identified subsequently over a three month period in 1994 were included in the study. It was found that age, shock, co-morbidity, diagnosis, major stigmata of recent haemorrhage, and rebleeding were all independent predictors of mortality when assessed using multiple logistic regression. A numerical score using these parameters has been developed that closely follows the predictions generated by logistical regression equations. When tested for general applicability in a second population, the scoring system was found to reproducibly predict mortality in each risk category

The Rockall risk score is a simple, validated predictive index that may serve as a useful clinical decision rule for assessing the risk of subsequent adverse outcomes in patients with UGI bleed. [51] An initial Rockall score based on clinical variables (age, shock, and co-morbidity) can range from 0 to 7 points. A complete score takes into account the endoscopic lesion categorization and stigmata of hemorrhage and can range from 0 to 11 points. For example, using the criteria described above, if a subject less than 60 years of age, with no co-morbidity, was found to have a Mallory-Weiss tear

and no stigmata of recent hemorrhage during endoscopy, he /she would have a total Rockall score of 0.

Table 3. Rockall Scoring system

Rockall Score				
Variable	0	1	2	3
Age (y)	<60	60-79	>80	
Shock	No Shock (Systolic BP >100 mm Hg; pulse <100 beats/min)	Tachycardia (Systolic BP >100 mm Hg; pulse >100 beats/min)	Hypotension (Systolic BP <100 mm Hg; pulse <100 beats/min)	
Co morbidity	None	-	Cardiac failure, Ischemic heart disease, any major co morbdity*	Renal failure, liver failure, disseminated malignancy
Diagnosis	Mallory-Weiss, no lesion or stigmata of recent hemorrhage	All other diagnosis	Malignancy of the upper GI tract	-
SRH	None or dark spot	-	Blood in upper GI tract, adherent clot, visible or spurting vessel	-
*Any major comorbidity would be defined as any other immediately unstable life threatening illness in addition to cardiac failure, IHD, renal/liver failure and cancer etc. (Rockall et al., 1996)				

The patients were classified into three risk groups, based on the Rockall score. Those with a score less than 3 fell into group A (low risk), score of 3-5 were placed in group B (moderate risk) and those with a score of 6 or more in group C (high risk).

For cases with a score of less than three, several studies suggest that rebleed occurred in less than 5% of patients and death occurred in less than 1% of patients [52,53,54] , but a score in excess of 8 is associated with a 41% mortality and rebleeding rate of 42.1%.

Rockall et al. noted that 45.4% of their patients belonged to the low risk group, 50.7% to the moderate risk and 3.9% to the high risk group and the percentage of rebleed progressively increased from the low risk (8.8%) to the high risk groups (17%) Barkun et al. based on data from the RUGBE database, found that 13% , 53% and 34% belonged to low, moderate and

high risk groups respectively, with a higher percentage, belonging to the high risk group, as compared to that noted by Rockall et al.

Overall rebleed rates of 13.8% were noted by Barkun et al.[1] Rockall et al found it to be 15.4% but Yavorski et al observed a much lower rebleed rate of 7.1%.

Comorbidities were noted by Rockall et al in 59.1%,and Yavorski et al in 50.9%.[9] and hemodynamic instability was noted by Rockall et al. in 11.2%.Yavorski et al.[9] noted that blood transfusions were instituted in 47.3% of their cases.

The Rockall system has been shown to represent an accurate and valid predictor of rebleeding and death. This score can be used to compare outcomes in audit and research and to calculate risk standardised mortality. This has the potential to result in a more appropriate management of subjects' conditions based on their assessed risk of complications following the initial UGI bleeding.

In addition, this risk score can identify 15% of all cases with acute upper gastrointestinal hemorrhage at the time of presentation and 26% of cases after endoscopy, who are at low risk of rebleeding and negligible risk of death and who might therefore be considered for early discharge or outpatient treatment with consequent saving of resources. Such risk assessment scores may be useful in triaging patients for either outpatient care or admission to a high dependency unit.

Among these studies, Sanders et al. [53] prospectively studied 325 patients admitted to a specialized hemorrhage unit over a 3-year period. The aim of their study was to assess the validity of the Rockall risk-scoring system

in predicting rebleeding and mortality in subgroups of patients with esophageal varices or peptic ulcers. The results of their study were comparable to those of Rockall's initial cohort in predicting rebleeding and death in patients with either ulcers or varices (scores of < 3 accounted for 29.4% of patients, of whom only 4.3% rebled and 0.1% died).

Enns et al.[55] noted that Rockall scoring system has a good discriminative ability and provides an acceptable tool to predict death, but performs poorly for endpoints of rebleeding and surgical procedures.

Vreeburg et al. [54] concluded that the risk scoring system developed by Rockall and coworkers is a clinically useful scoring system for stratifying patients with acute UGIB into high and low risk categories for mortality. For the prediction of rebleeding, however, the performance of this scoring system was unsatisfactory.

Dulai et al.[51] conducted a retrospective study to accurately risk stratify patients by using the Rockall score. Their findings suggested that a significant number of all patients hospitalized with acute UGIB are at low risk of adverse outcomes related to their hemorrhage episodes.

Oei and colleagues [52] evaluated and compared the incidence of low-risk UGIB admissions, adverse outcomes, and the levels of healthcare resource use in a community hospital and a university hospital. The data from their study confirmed the low rate of morbidity and mortality in both practice settings, suggesting that downgrading the site of initial admission for low-risk patients with early discharge could conserve healthcare resources without compromising patient safety.

In a study by Akash et al. [56] from Chennai, Rockall score was found to correlate well with clinical outcome including rebleeding and mortality.

These studies demonstrate that patients with a low Rockall score can be managed safely as outpatients, or with limited admission and early discharge, without adversely influencing patient outcomes and with considerable resource savings.

Blatchford and colleagues[42] developed and tested a simple scoring system to identify patients at high or low risk of requiring hospital admission and aggressive treatment to control gastrointestinal bleeding. They studied 1748 patients admitted with upper GI bleed and used logistic regression in the derivation of risk score. This scoring system does not require endoscopic evaluation to aid risk stratification

Table 4. Blatchford scoring system

Risk marker	*Score component value*	*Risk marker*	*Score component value*
Blood urea nitrogen--mg per dL (mmol per L)		**Systolic blood pressure-mm Hg**	
≥18.2 and <22.4 (≥6.5 and <8.0)	2	100 to 109	1
≥22.4 and <28.0 (≥8.0 and <10.0)	3	90 to 99	2
≥28.0 and <70.0 (≥10.0 and <25.0)	4	<90	3
≥70.0 (≥25)	6	**Other markers**	
Hemoglobin in men--g per dL (g per L)		**Pulse >=100 per minute**	1
≥12.0 and <13.0 (≥120 and <130)	1	**Presentation with melena**	1
≥10.0 and <12.0 (≥100 and <120)	3	**Presentation with syncope**	2
<10.0 (<100)	6	**Hepatic disease**	2
Hemoglobin in women--g per dL (g per L)		**Cardiac failure**	2
≥10.0 and <12.0 (≥100 and <120)	1		
<10.0 (<100)	6		

The Baylor Group developed and validated the Baylor Bleeding Score to identify patients who might require early surgical intervention.[57] By assessing simple pre-endoscopic (age; number and severity of concurrent

medical illnesses) and post-endoscopic parameters (site and stigmata of bleeding ulcers), Saeed et al. showed that this scoring system might be able to predict patients at risk of rebleeding after successful endoscopic therapy of bleeding ulcers.

APACHE II scoring system has been used in measuring the severity of acute illness and Schein and Gecelter [58] noted that among 96 patients operated for bleeding peptic ulcers, none of the patients with a score of less than 11 died, whereas the mortality in those who scored more than 10 was 22%, thus indicating its usefulness in predicting outcome in these patients.

ACUTE NON-VARICEAL UPPER GASTROINTESTINAL BLEED

Pharmacological management

If the gastric pH is maintained above 6 (by infusional PPI), platelet aggregation is optimized and fibrinolysis relatively inhibited, thereby potentially improving the likelihood of clot stability at the ulcer site. Individual trials of H2 receptor antagonists (H2RA) have generally failed to demonstrate a clinical benefit in UGIB.

Several studies have evaluated intravenous proton pump inhibitors (PPI) for non-variceal UGIB; in the usual intravenous 80mg bolus dose followed by a continuous infusion of 8mg/hour for up to 72 hours and this has now shown a benefit in terms of re-bleeding, need for surgery and mortality.[59] Proton pump inhibitors have been advocated, by ASGE, prior to endoscopy, for bleeding peptic ulcers and in suspected peptic ulcer bleeds.

Bolus administration of intravenous erythromycin prior to endoscopy has been shown to clear the stomach of blood, increases the likelihood of successful haemostasis and reduces the need for subsequent interventions.[60] The usefulness of somatostatin and its analogue, octreotide, is a matter of debate.[61]

TREATMENT OF ULCERS IN NSAID USERS

Proton pump inhibitors are superior to H2RAs and misoprostol for healing NSAID ulcers in the setting of continued NSAID use. In the "Acid Suppression Trial: Ranitidine versus Omeprazole for NSAID Associated Ulcer Treatment" (ASTRONAUT) study, 541 patients with ulcers or extensive erosions were randomized to omeprazole 20 or 40 mg or ranitidine 150 mg twice daily. After 8 weeks of treatment, the rates of healing in all types of lesions were higher in those treated with omeprazole compared with ranitidine. The higher dose of proton pump inhibitor was not superior to lower dose [62] and similar data exist for other proton pump inhibitors.[63] In the "Omeprazole versus Misoprostol for NSAID-Induced Ulcer Management" (OMNIUM) study, in which 900 NSAID using patients with ulcers or extensive erosions were randomized to receive misoprostol 200 µg 4 times a day or omeprazole 20 or 40 mg once daily for 8 weeks, gastric ulcer healing was significantly more frequent on 20 mg of omeprazole compared with misoprostol. The rates of duodenal ulcer healing were also significantly higher in the groups given omeprazole 20 or 40 mg compared with misoprostol.[64]

If the patient can discontinue the NSAID, all forms of anti-ulcer therapy work effectively. The standard of care remains that all patients with

peptic ulcer disease, whether taking NSAIDs or not, undergo testing for and treatment of *H. pylori* infection.

Treatment of H. pylori infection

Two Antibiotics Plus One Adjunctive Agent (Triple Therapy)

Triple therapy with either bismuth or a PPI combined with two antibiotics is now the most widely used regimen. Therapy with bismuth, metronidazole, and tetracycline ("traditional" triple therapy) produces very good cure rates, especially with organisms sensitive to metronidazole. Substitution of clarithromycin for metronidazole gives similar results.[65] The most popular triple therapy combines a PPI with two of these three antimicrobials: amoxicillin, metronidazole and clarithromycin.

Two Antibiotics Plus Two Adjunctive Agents (Quadruple Therapy)

This consists of metronidazole (500 mg three times daily), tetracycline (500 mg three or 4 times daily), bismuth subsalicylate or subcitrate (three or four times daily) and a PPI twice daily.

THERAPEUTIC STRATEGY

The most effective regimens to cure *H. pylori* infection are combinations of two antibiotics and adjunctive agents taken for 14 days. The most effective and best tolerated combination seems to be a twice-a-day combination of 1000 mg of amoxicillin and 500 mg of clarithromycin (PPI + AC) or 500 mg of metronidazole and either 250 or 500 mg of clarithromycin (PPI + MC).

ENDOSCOPIC THERAPY

Endoscopic therapy has been shown to improve outcome in nonvariceal haemorrhage. In a recent meta-analysis of 30 randomized trials involving more than 2000 patients, endoscopic therapy reduced rates of further bleeding, need for urgent surgery and mortality. [66]

Endoscopic therapy is indicated when there are major stigmata of recent haemorrhage (SRH). There is little doubt that Forrest IA, IB and IIA ulcers should have endoscopic hemostasis. [67] Patients with an adherent clot may also constitute a high-risk group. Up to one-third of blood clots covering an ulcer can be removed to reveal major stigmata of recent hemorrhage. Current opinion favours the displacement of the clot by irrigation or mechanical removal, followed by endoscopic hemostasis of any underlying visible vessel. Forrest IIC and III ulcers may be managed conservatively and discharged early.

Several endoscopic therapies have been described in the treatment of actively bleeding Mallory-Weiss lesions and have included endoscopic electrocoagulation, epinephrine injection or heater probe cauterization.

ENDOSCOPIC TREATMENT FOR NON-VARICEAL UPPER GASTROINTESTINAL BLEEDING- VARIOUS MODALITIES [51]

1. **Thermal**
    a. Heater probe
    b. Multipolar electrocoagulation (BICAP, Gold Probe)
    c. Argon plasma coagulation
    d. Laser

2. **Injection**
    a. Adrenaline (1:10000)
    b. Procoagulants(fibrin glue,human thrombin)
    c. Sclerosants (ethanolamine, 1% polidocanol)
    d. Alcohol (98%)
3. **Mechanical**
    a. Clips
    b. Band Ligation
    c. Endoloops
    d. Staples
    e. Sutures
4. **Combination therapy**
    a. Injection plus thermal therapy
    b. Injection plus mechanical therapy

Recent focus has been directed towards combination therapies and mechanical means of homeostasis and it has been suggested as the recommended line of management by international and ASGE guidelines.

Injection therapy

Injection of dilute (1:10,000) adrenaline in 1 ml aliquots around the bleeding points results in hemostasis in upto 100% of patients with bleeding peptic ulcers, probably by a combination of vascular tamponade and vasoconstriction, with a concomitant reduction in re-bleeding rates from 40% to 15%.[68] The dose required is variable but larger volumes (13-20ml vs. 5-10ml) in high risk patients (Forrest type I or IIa lesions) results in less rebleeding (15.4% vs. 30.8%). Although injection with adrenaline is successful

in achieving initial hemostasis, 15-36% of patients were found to have rebleed.[69] Sclerosants such as ethanol, polidocanol and ethanolamine are as effective as adrenaline but carry more risk.

Thermal techniques

Thermal hemostasis is achieved by compression of the artery during heating (coaption) and/or the effect of heat on tissue.

Non-contact thermal techniques currently available are Argon Plasma Coagulation (APC) and laser (Nd:YAG). APC involves conduction of a high frequency electrical current through a beam of ionized argon gas, resulting in superficial tissue damage and coagulation. A prospective observational study of APC in 254 patients with non-variceal UGIB revealed initial hemostasis rates of 75.9% and re-bleeding rates of 5.7%.[16] Due to technical constraints of the technique, laser therapy is not routinely used in the management of non-variceal UGIB.

In contrast to APC and laser, Bipolar Electrocoagulation (BPE) and Heater Probe Thermocoagulation (HPT) use thermal contact to achieve haemostasis by compression of the vessel, Combination therapy with HPT and adrenaline in the treatment of actively bleeding peptic ulcers resulted in haemostasis in up to 98.6%, with re-bleeding in 8.2% [70] although added benefit is confined to high risk lesions. The risks associated with application of heat to bleeding lesions are due to the requirement for tissue contact, lack of control of depth of injury and difficulty in treating multiple or diffuse lesions.

Mechanical hemostasis

Mechanical hemostasis with endoloops or clips , has an increasing role in the control of non-variceal UGIB. Endoclips are deployed on a visible

vessel to achieve vascular compression and can achieve hemostasis in up to 100% of cases. Comparative studies suggest lower rebleeding rates than adrenaline injection.[71] Hemoclips can be technically difficult to apply if the ulcer is relatively inaccessible, for instance high on the gastric lesser curve or on the posterior duodenal wall.

Endoscopic band ligation (EBL) is currently technically easier to use than endoclips and has been shown to be safe and effective for control of small lesions in a small series of acute peptic ulcer bleeding [72] and with bleeding due to Dieulafoy's lesions. Newer techniques under evaluation include endoscopic suturing and cryotherapy .[73]

Vreeburg et al. in their series, noted endoscopic intervention rates of 21% , with injection therapy being instituted in as many as 74% with bleeding peptic ulcers.

"SECOND-LOOK" ENDOSCOPY AND ENDOSCOPIC RE-TREATMENT

Routine "second look" endoscopy, in the absence of established rebleeding or patient instability, has gone out of vogue after studies showed no benefit with regard to clinically significant outcomes for unselected patient populations, although there may be a role in high risk patients. Repeat therapeutic endoscopy may be indicated (depending on local endoscopic and surgical expertise) if there is clinical evidence of re-bleeding[74] or if the initial therapeutic procedure was unsuccessful or partially successful.

Surgical therapy in non variceal bleed

Pharmacologic and endoscopic approaches have progressively curtailed the use of operative therapy for PUD. Elective surgery is now rarely indicated and emergency operations are much less common. Vagotomy and

drainage procedures are technically simple but are associated with higher ulcer recurrence rates. Vagotomy and resection approaches offer lower ulcer recurrences but are associated with considerable mortality and morbidity.[75, 76] Gastric devascularisation has been tried as a salvage procedure for hemorrhagic gastritis.

ACUTE VARICEAL UPPER GI BLEED[1]

Resuscitation

A patient with variceal hemorrhage requires immediate stabilization. During resuscitation, coagulopathy should be corrected with fresh frozen plasma, vitamin K and platelet transfusions, if required.

Pharmacotherapy

Pharmacologic efforts to treat variceal bleeding have focused on diminishing portal blood pressure by shunting blood away from the mesentery through the use of smooth muscle constrictors. Vasopressin causes splanchnic vasoconstriction and intravenous infusion causes decreased portal blood pressure with an increase in systemic arterial pressure and a decrease in heart rate. Terlipressin is a long-acting analogue of vasopressin that also reduces portal blood flow through splanchnic vasoconstriction. It has a slightly better safety profile and can be dosed at 4–6-hour intervals rather than by continuous infusion. For both agents, because the vasoconstriction is nonspecific, mesenteric or cardiac ischemia can occur.

Nitrate preparations

By causing venodilation, nitrates reduce systemic blood pressure and mildly decrease portal blood pressure. In studies that combined nitrates with

vasopressin, bleeding control was improved and toxicity was less compared with vasopressin alone. [77]

Somatostatin and analogues

The synthetic analogue of somatostatin, octreotide is thought to have three principal mechanisms in variceal bleeding. It blocks the increase in hepatic venous pressure, causes splanchnic vasoconstriction and downregulates enteric secretion and motility. Its low toxicity profile has made it a popular empiric choice for suspected portal hypertensive bleeding. In trials of acute variceal bleeding, it was more or at least as effective as vasopressin but with fewer adverse effects.[78]

Endoscopic Therapy

It should be performed in an intensive care unit after adequate volume resuscitation. Endotracheal intubation with mechanical ventilation should be considered in any patient with active hematemesis or a decreased level of consciousness in order to protect the airway and to minimize the chance of aspiration.

The mainstay of endoscopic therapy for bleeding oesophageal varices is injectable vascular sclerosants. There are several types of sclerosants including morrhuate, tetradecyl sulfate and ethanolamine. They can be injected intravariceally or paravariceally.

Endoscopic sclerotherapy (EST) produces hemostasis by injuring endothelium and provoking variceal thrombosis and through a pressure effect from thrombus formation in an adjacent blood vessel.[79] Total obliteration of varices usually requires multiple endoscopic sessions. Sclerotherapy can be

complicated by chest pain, fever, pleural effusion and dysphagia. Esophageal ulceration with late stricture formation, perforation and bacteremia are other possible sequelae.

More recently, endoscopic variceal ligation (EVL) has emerged as an effective treatment for esophageal varices. Using a transparent cylinder attached to the end of the endoscope, a varix is suctioned into the cylinder, and a rubber band is deployed around the varix, causing hemostasis, thrombosis, and sloughing of the variceal column. EVL may be technically more difficult in an actively bleeding patient because visualization of the varix is recommended before suction is applied.

In a comparison of EVL and sclerotherapy for treatment of active bleeding in cirrhotic patients, EVL was more successful for control of spurting varices. Bleeding ceased for at least 3 days in 97% of the EVL patients but in only 76% of the sclerotherapy patients. In the same study, EVL patients also required fewer blood transfusions and had fewer complications (5% vs. 29%) and lower mortality than patients treated with sclerotherapy.[80] In a recent randomized controlled trial, EVL alone was compared with EVL and adjuvant sclerotherapy of varices that were too small to be eradicated by banding. Although complication rates and recurrent bleeding rates were similar between the two groups, the patients who received adjuvant sclerotherapy had a significantly lower rate of variceal recurrence. At 1 year, the likelihood of variceal recurrence was 45% among patients who received only EVL as compared to 24% for those who also received sclerotherapy.[81]

These studies suggest that optimal results may be seen from a combination of endoscopic therapies to control bleeding and sequentially eradicate varices to prevent rebleeding.

When esophageal varices show endoscopic stigmata of recent hemorrhage or when there is a high clinical suspicion that variceal bleeding is responsible for the patient's hemorrhage, endoscopic variceal ligation should be performed at 1–2 week intervals until the varices are obliterated. Follow-up endoscopy would be performed every 3–6 months thereafter to rule out variceal recurrence.

EVL has replaced sclerotherapy as the standard endoscopic treatment to prevent rebleeding because EVL obliterates varices in fewer treatment sessions with a lower rate of rebleeding and lower mortality.[82] A Japanese study that compared EVL with sclerotherapy for treatment of variceal bleeding in 101 patients found that hemostasis could be achieved in all patients of both treatment groups and that obliteration was approximately 90% in both groups. However, the rate of rebleeding was 40% in the sclerotherapy group and only 29% in the EVL patients.

On an average, EVL treatments were completed in 2.1 sessions versus 3.7 sessions for sclerotherapy. The most common complications, rebleeding and intramural hematomas, were seen less frequently in patients who received EVL.[83]

Active bleeding from gastric varices or portal hypertensive gastropathy may be difficult to treat endoscopically, though heater or bipolar probe and argon plasma coagulation have been tried in acute bleeding from portal

hypertensive gastropathy and sclerosant and glue injection as well as banding have been tried for gastric varices.

Surgical and Angiographic Shunts

When portal hypertensive bleeding (oesophageal or gastric varices or portal hypertensive gastropathy) cannot be controlled with medical or endoscopic therapy, surgical shunts and angiographic portosystemic shunts (Transjugular Intrahepatic Portosystemic Shunting) should be considered.

There are several surgical shunt options: portocaval, mesocaval, and splenorenal shunts. An additional surgical option to control variceal hemorrhage is oesophageal transection.

CHAPTER 4

MATERIALS AND METHODS

Four hundred and six consecutive patients with Upper GI bleed, referred for upper GI endoscopy to the endoscopic unit of Govt. General Hospital, Chennai were included in the study.

Design of the study - Prospective cross sectional study

Period of study - One year

Ethical clearance - Obtained

Consent - Informed consent from all the patients

Patient selection

Inclusion criteria

Patients with upper GI bleed (hematemesis, melena or hematochezia with bloody nasogastric aspirate)

The patients who fulfilled the above mentioned criteria and did not have any contraindications for endoscopy and were willing for undergoing upper GI endoscopy were enrolled in the study.

Exclusion criteria

1. Comatose patients
2. Patients with stage 3 and 4 Hepatic encephalopathy
3. Myocardial infarction
4. Perforated viscus
5. Lack of willingness to undergo UGI endoscopy

Protocol

1. All patients who met the above criteria were included in the study

The following were noted in each patient

1. Age
2. Gender
3. Presentation- hematemesis, melena, hematochezia
4. Severity of bleed-minor, moderate or massive
    - Minor bleed-no hemodynamic instability, <10% blood loss
    - Moderate bleed - postural hypotension and orthostatic tachycardia, 10-20% intravascular volume loss
    - Massive bleed-shock (resting hypotension) - 20-25% intravascular volume loss
5. Associated factors-Alcohol use, smoking, use of non-steroidal anti-inflammatory drugs (NSAID), corrosive ingestion and past history of upper GI bleed
6. Comorbid conditions –Ischemic heart disease, congestive cardiac failure, renal failure, liver disease, disseminated malignancy
7. Physical examination and documentation of tachycardia and hypotension and the clinical diagnosis
8. Requirement for blood transfusion

Endoscopy

Upper GI endoscopy was performed in all patients, findings documented and requirement and mode of endoscopic therapy to control bleeding in indicated cases, noted.

Early endoscopy was defined as endoscopy performed within 24 hours of seeking medical care.

Peptic ulcers were classified based on the Forrest classification and varices by the classification proposed by Sarin et al. The grading of oesophageal varices was taken as follows: small and straight (grade I); tortuous and occupying less than one third of the esophageal lumen (grade II); or large and occupying more than one third of the esophageal lumen (grade III).

Rockall score was calculated in all patients based on the following criteria

- Age
- Shock (assessed from pulse rate and blood pressure)
- Comorbid conditions (cardiac, renal, liver, others)
- Endoscopic stigmata of recent bleed
- Endoscopic diagnosis

On summing up different levels of a point grading system, scores ranging from 0 to 11 were obtained.

Rebleed was defined as fresh hematemesis or melena associated with the development of shock or a fall in hemoglobin concentration of at least 2 g/dl in 2 hours.

The rebleed, requirement of blood transfusions and mortality were documented and the efficacy of Rockall score as a predictor of rebleed was analyzed.

The concordance between the initial clinical diagnosis and endoscopic findings were recorded.

Statistical analysis -Student's t test, Chi-square tests and Fisher's exact test as appropriate were used for comparison between groups. A P value of 0.05 or less was regarded as significant.

CHAPTER 5

RESULTS

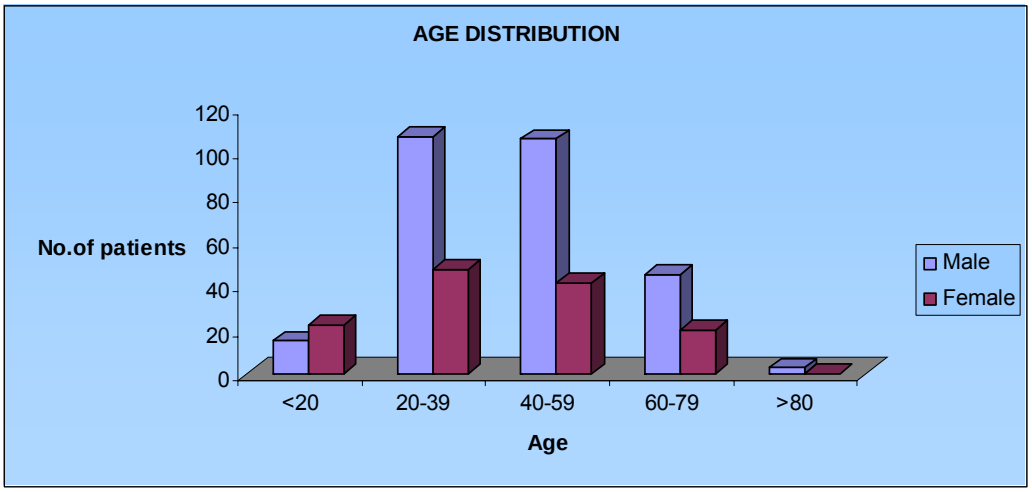
A total number of 406 patients were studied.(male-276 (68%),female-130 (32%) with an range of 12 – 84 years (Mean 40.8 years , SD-16.3 years).

The cases comprised 0.53% of the hospital admissions and 13.4% of upper GI endoscopies performed during the time duration of study.

Table 5.Age Distribution

Age group	Male	Female	No. of cases
<20	15	22	37
20-39	107	47	154
40-59	106	41	147
60-79	45	20	65
>80	3	0	3
Total	276(68%)	130(32%)	406

Fig 2.Age distribution



Most of the patients- 107 (25.8%) were observed to fall into the 20-39 years age group

Presentation

The majority of patients presented as hematemesis.

Table 6.Mode of presentation

Age	Hematemesis	Melena	H+M	Hematochezia	Total
<20	28	3	6		37
20-39	94	15	43	2	154
40-59	73	26	47	1	147
60-79	36	8	21	0	65
>80	3	0	0	0	3
total	234	52	117	3	406

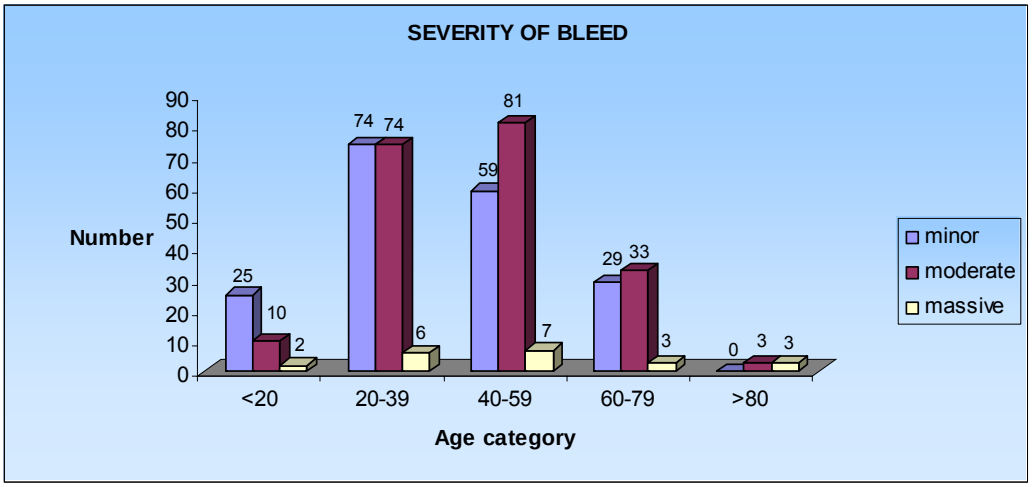
Severity of bleed

The majority of patients presented with moderate bleed, and only 18(4.4%) presented with massive bleed.

Table 7. Severity of bleed

Age	Minor	Moderate	Massive	Total
<20	25	10	2	37
20-39	74	74	6	154
40-59	59	81	7	147
60-79	29	33	3	65
>80	0	3	0	3
Total	187	201	18	406

Fig 3.Severity of bleed

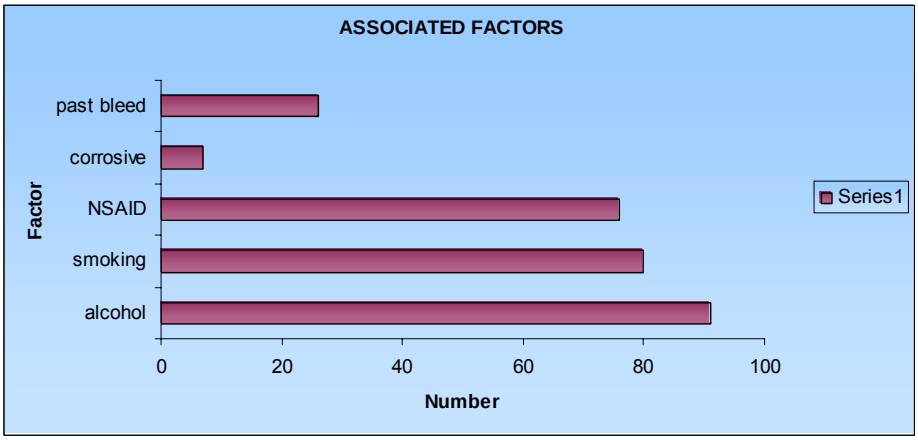


Associated factors

Table 8. Associated factors

Age	Alcohol	Smoking	NSAID	Corrosive	Past bleed
<20	0	5	3	3	4
20-39	31	31	20	3	11
40-59	51	32	29	1	10
60-79	9	11	23	0	0
>80	0	1	1	0	1
Total	91	80	76	7	26

Fig4.Associated.factors



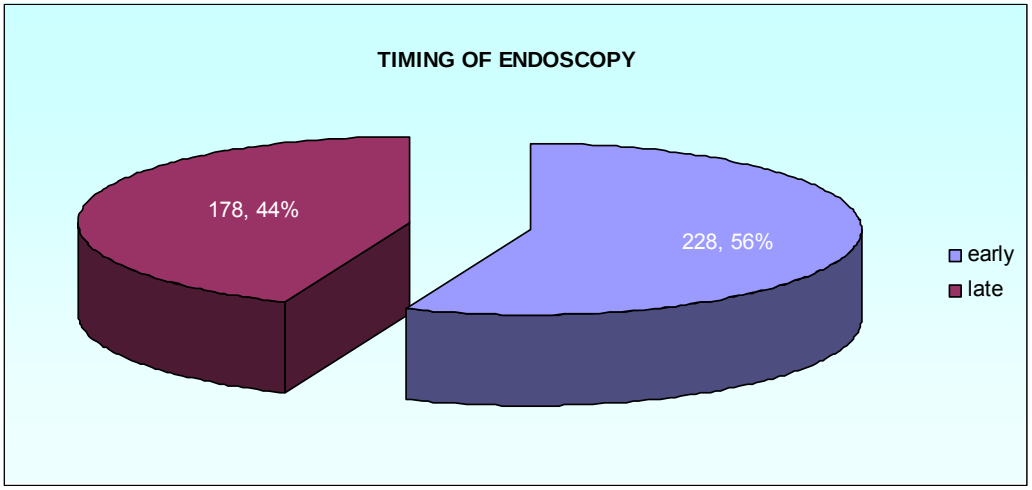
Alcohol, smoking and NSAID use were found to be the major associated factors.

The major comorbidities included cardiac problems including ischemic heart disease and congestive cardiac failure in 18 patients,renal failure in 4 patients and liver disease in 70 patients. Other comorbidities included COPD, diabetes mellitus and systemic hypertension.

Timing of Upper GI endoscopy

228 cases (56.2%) were subjected to Upper GI endoscopy to ascertain causes of bleed within 24 hours(early) and 178(43.1%) cases after 24 hours (late)

Fig 5. Timing of upper GI endoscopy



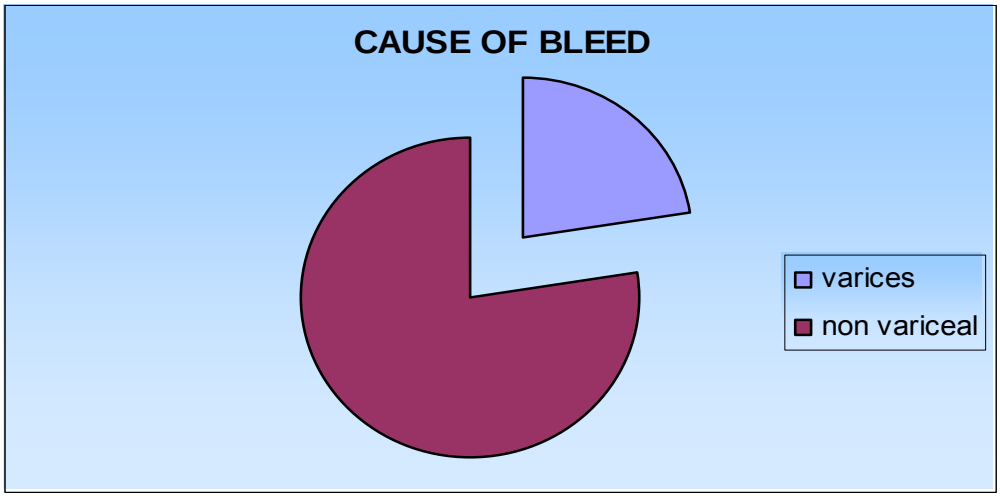
Endoscopic findings

Non variceal causes predominated and were the most common sources of bleed in all age categories. The upper GI endoscopy was non contributory to a diagnosis in 37 cases.

Table 9. Cause of bleed

Varices	Non Variceal
91(22.4%)	315(77.6%)

Fig 6.Cause of bleed



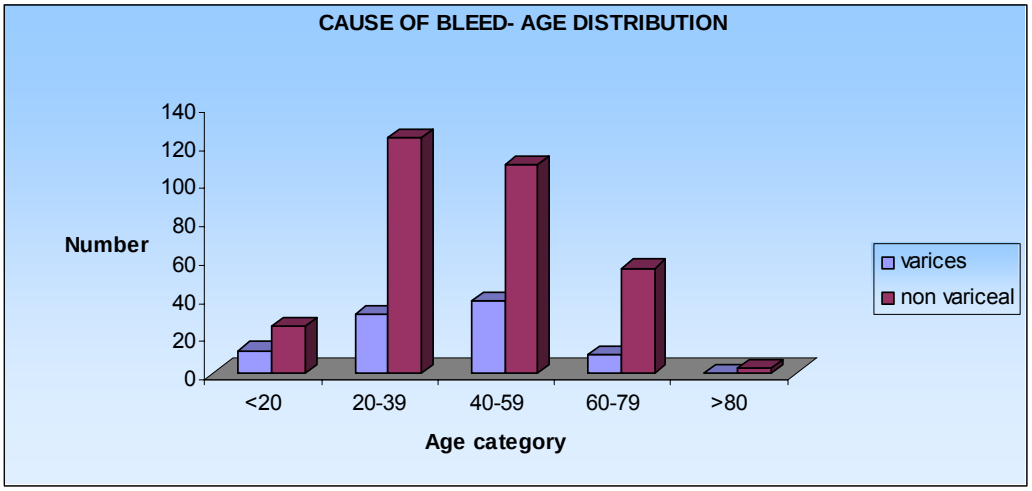
Causes of bleed- age distribution

Table 10.Causes of bleed- age distribution

Age	Varices	Nonvariceal	Total
<20	12	25	37
20-39	31	123	154
40-59	38	109	147
60-79	10	55	65
>80	0	3	3
Total	91	315	406

Nonvariceal causes of UGI bleed predominated in all age groups.

Fig 7. Cause of bleed- age distribution



Nonvariceal causes

Table 11.Nonvariceal causes

Age	Duodenal ulcer	Gastric ulcer	MW tear	Gastritis	Duodenitis	Esophagitis	Malignancy	Angiodysplasia
<20	4	2	0	13	11	5	1	1
20-39	23	5	1	69	38	29	2	3
40-59	16	16	0	62	41	24	8	1
60-79	8	5	1	29	16	11	5	0
>80	1	0	0	1	1	2	0	0
Total	52	28	2	174	107	71	16	5

Since some subjects had more than one lesion, (eg. duodenal ulcer with gastritis), the sum total of all lesions in the nonvariceal category exceeded the total number of patients (315) with nonvariceal bleed.

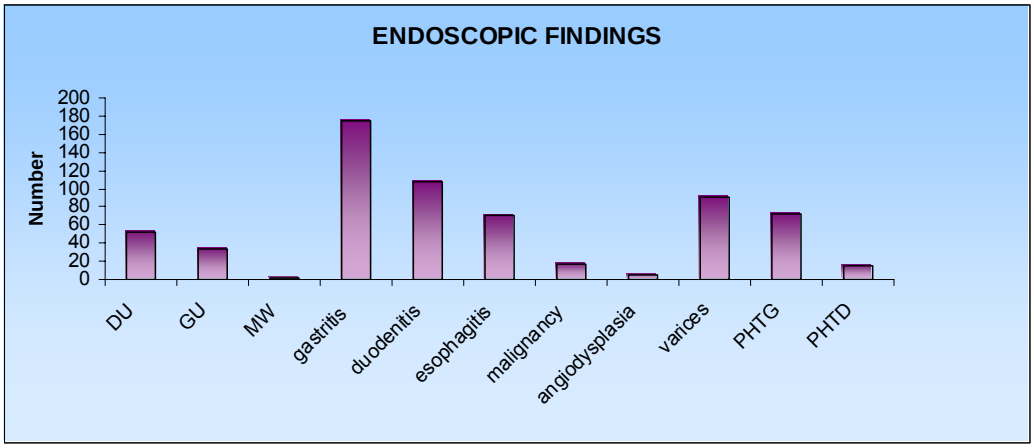
Variceal causes

Table 12. Variceal causes

Age	Oeso Varices	GOV1	GOV2	IGV1	IGV2	Varices
<20	5	1	3	3	0	12
20-39	16	5	9	1	0	31
40-59	21	4	11	2	0	38
60-79	2	2	6	0	0	10
>80	0	0	0	0	0	0
Total	44	12	29	6	0	91

Portal hypertensive gastropathy and duodenopathy were found in 72 and 15 subjects.

Fig8.Diagnosis



Endoscopy and clinical diagnosis

Endoscopic findings were more in concordance with clinical diagnosis in MW tear (90%), duodenal ucer (88%), varices (84%) and gastritis (80%) compared to malignancy (60%) and gastric ulcer(52%)

ENDOTHERAPY

Varices

Table 13.Grading of varices and endotherapy

Varices	Number of cases	Endotherapy done
Grade I	8	-
Grade 2	31	30
Grade 3	46	45
IGV	6	-
Total	91	75

Peptic ulcers

Table 14.Forrest classification and endotherapy

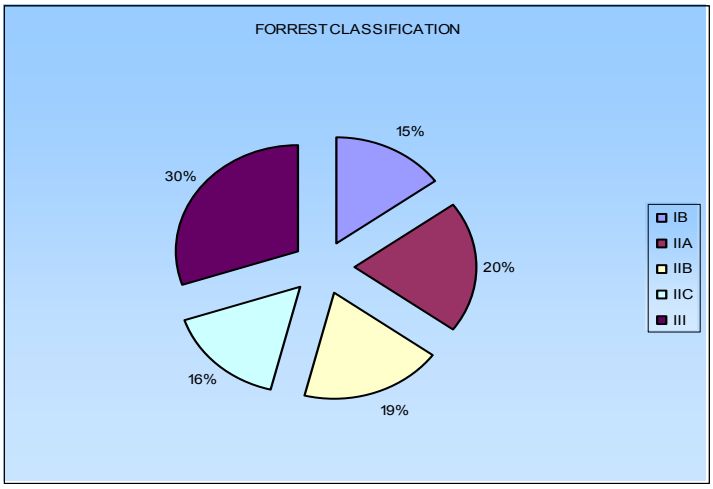
Forrest class	Number of cases	Endotherapy
IB	12	11
IIA	16	15
IIB	15	14
IIC	13	-
III	24	-
Total	80	40

The peptic ulcers that were found to have an active ooze, visible vessel or adherent clot (Forrest class IB, IIA, IIB), were subjected to endotherapy.

Of the 171 cases with varices and peptic ulcers, endotherapy was done in 115 (50% of peptic ulcers and 82.4% of variceal bleed) cases.

Adrenaline injection was performed in 40 cases; endoscopic sclerotherapy was done in 40 cases and endoscopic variceal ligation in 35 cases

Fig 9. Forrest classification of peptic ulcers



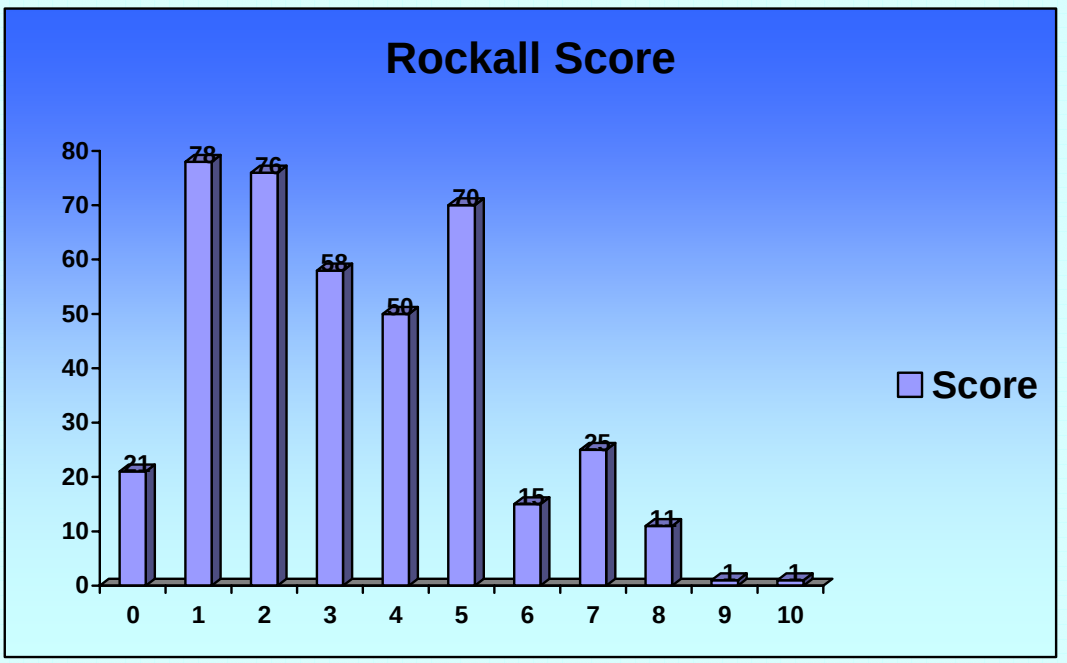
Rockall score

The mean Rockall score was calculated in all patients based on the age, hemodynamic status, comorbidity, endoscopic diagnosis and SRH. The mean Rockall score was 3.3(S.D 1.7, range 0 – 10)

Table 15.Rockall Score- distribution

Score	No. Of Patients
0	21
1	78
2	76
3	58
4	50
5	70
6	15
7	25
8	11
9	1
10	1

Fig10.RockallScore



The patients were classified into three risk groups, based on the Rockall score. Those with a score less than 3, into group A (low risk), score of 3-5 into group B (moderate risk) and those with a score of 6 or more into group C (high risk). Most patients were observed to belong to the first two groups. The mean Rockall score in the low risk group was 1.3, that of the moderate risk group was 4.1 and that of the high risk group was 7.

The distribution of cases in all the three groups with respect to mean age, gender, comorbidity, hemodynamic status and the endoscopic diagnosis, was recorded.

Rockall Score- risk groups

Table 16.Risk groups- age distribution

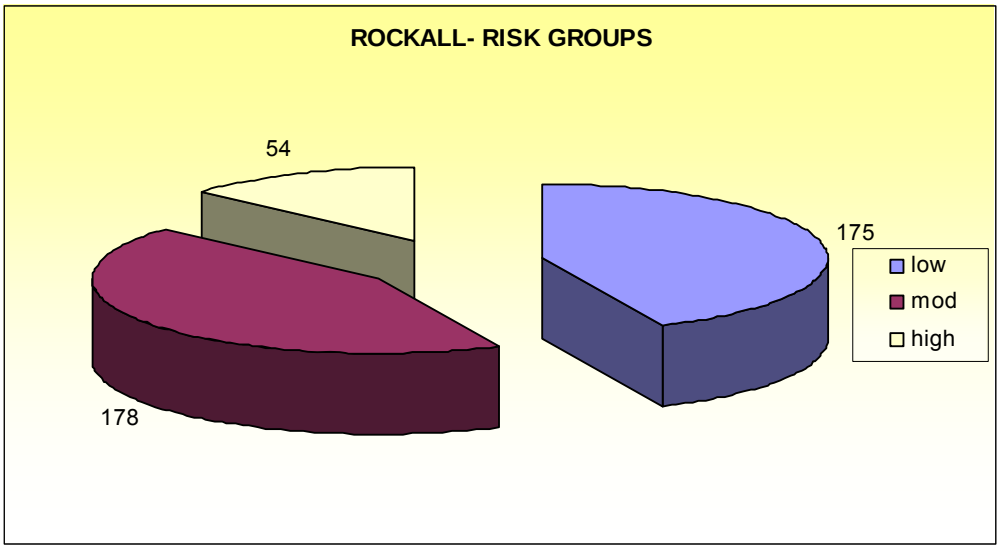
Age	Low risk(A)	Mod risk(B)	High risk(C)	Total
<20	20	16	1	37
20-39	79	59	16	154
40-59	62	62	23	147
60-79	14	40	11	65
>80	0	1	2	3
Total	175	178	53	406

In the the high risk group, the number of patients aged 60 years or more was not found to be statistically higher(P=0.11)

Table 17. Risk groups- variables

Variable	Low Risk(A)	Mod Risk(B)	High Risk(C)
N	175	178	53
Mean Age	36.5	43	47
Male	113	122	41
Comorbidity	5	91	45
Tachycardia	68	148	53
Hypotension	0	82	45
No Lesion	36	1	0

Fig .11.Rockall Score- risk groups



Rockall Score and cause of bleed

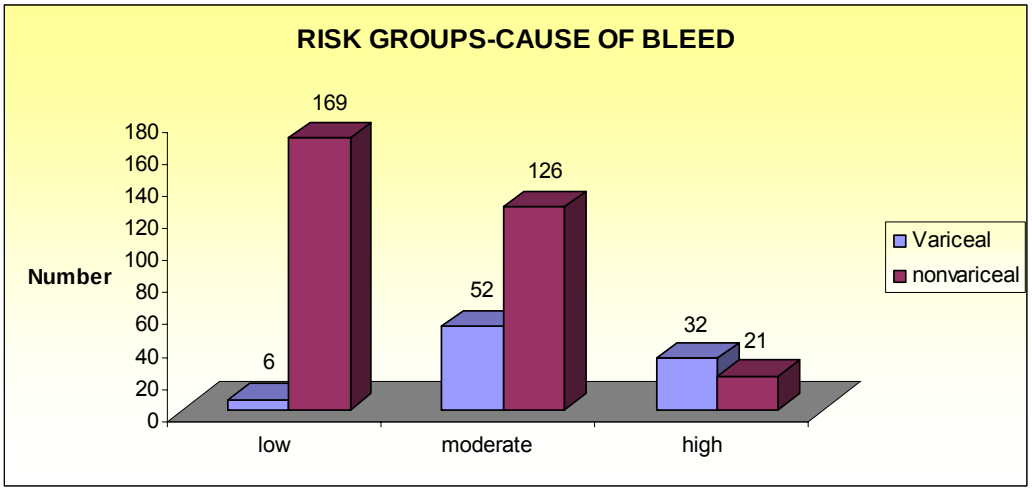
Variceal bleed was observed in a higher proportion in the moderate and high risk groups.

Table 18.Risk groups- cause of bleed

Cause	Low	Moderate	High
Varices	6	52	32
Nonvariceal	169	126	21

In the high risk group, the number of bleeds due to variceal causes were significantly higher as compared to the low risk group.(P value<0.001)

Fig 12.Risk groups- cause of bleed



Rockall Score and outcome

The need for packed red cell transfusion and the number of patients who had rebleed were noted, along with mortality.

Table 19.Rockall Score and outcome

Variable	Low Risk	Mod Risk	High Risk	Total
PRBC	8	72	41	121
Rebleed	4	28	32	64
Mortality	0	0	3	3

The requirement of blood transfusion and the incidence of rebleed was higher with moderate and high risk groups.

Table 20.Risk groups and outcome- comparison

	Low vs. mod risk P value	Low vs. High risk P value
PRBC transfusion reqt	<0.001	<0.001
Rebleed	<0.001	<0.001
Mortality	1	0.01

The requirement of blood transfusion was significantly higher in the moderate and high risk groups as compared to the low risk groups, (both P values less than 0.001) , as was the incidence of rebleed which was more in the moderate and high risk groups as against the low risk group.(P<0.001 in both)

There was no mortality in the moderate and low risk groups. There were 3 deaths in the third group which was statistically significant as compared to the low risk group and moderate risk group. (P value =0.01)

It was noted that in the 64 cases who had rebleed, 44(68.8%)had nonvariceal causes [peptic ulcer- 21(Forrest IB(6), IIA(7), IIB(8)), gastroduodenitis-18, oesophagitis- 3, gastric malignancy-2]and the rest were variceal (grade III varices-12, grade II varices-8) .The rebleed was not found to be significantly higher for variceal causes (P value = 0.7), though a higher proportion of varices were found to have a rebleed..

Of the patients who had rebleed, 9 had received prior EST and 7 had received EVL . In peptic ulcers with rebleed, 18 of the 21 cases with rebleed had received prior injection therapy with adrenaline (Forrest IB-5, IIA-6,IIB-7). Following the rebleed, 11 patients with varices were given EST and 9 patients were subjected to band ligation of varices. Adrenaline was injected locally to control rebleed in all the rebleeding peptic ulcers.

CHAPTER 6

DISCUSSION

The clinical and endoscopic profile of the four hundred and six patients presenting to the endoscopy unit of the institution were analysed. Age, gender, severity of bleed, mode of presentation, etiology and associated factors were documented, along with the documentation of Rockall score, requirement of blood transfusions, rebleed and endotherapy instituted.

The Rockall score has been validated by several studies, for predicting rebleed and mortality. In this study, along with the determination of the Rockall score, the subjects were divided into three risk groups. The available data is compared with contemporary literature and utility of Rockall score in risk stratification, is evaluated.

Age distribution

The mean age was 40.8 years (S.D 16.3 years) with an age range of 12-84 years. It was lower than that observed in the RUGBE (Canadian Registry on Upper Gastrointestinal Bleeding and Endoscopy Database of 1869 patients) study by Barkun et al.[11], Rockall and Logan[8] et al. and Yavorski et al.[9]

Table 21.Age distribution- comparison

	Barkun et al[11]	Rockall et al[8]	Yavorski et al[9]	Present study
Mean age	66	66	52.06	40.8
Age Range	7-105	16-103	1-99	12-84
Total number	1869	2332	3294	406

The majority of patients in the present study fell into the age group of 20-39 years -157(38.7%). Only 3 subjects (0.7%) were above 80 years, as compared to 634 (27.2%) noted by Rockall et al, who concluded that the incidence of bleed significantly increased with age.

Longstreth and colleagues, in their series, noted that 47% of their patients were above 60 years of age, as compared to the present study where it was noted to be only 9.9%.

Gender distribution

Male predilection (68%) was noted in this study, in concordance with the RUGBE database, where 62% were males and that noted by Rockall et al. (57% males). Longstreth et al also had noted a male predilection of 67.9%.

Presentation

Majority of patients patients-351(86.5%) had hematemesis as a presenting feature and 169(41.6%) had melena, as compared to RUGBE data[11] where hematemesis was noted in 58% of patients and melena in 69%, and that observed by Longstreth et al [10] who noted that 33% of their patients had hematemesis and 81% had melena.

Hematochezia was noted in 3(0.7%), which was much lower than that noted by Barkun et al(15%) and Laine et al (5%).[13]

Associated factors

Longstreth et al noted history of NSAID use in 53% and alcohol use in 3% of patients in their series. The present study noted a much higher percentage of patients with alcohol use- 91(22.4%) , though NSAID use was noted only in

76(18.7%) of patients. RUGBE data showed a previous documented UGIB in 19.5% of patients, whereas in the present study, it was noted to be 6.4%.

Timing of endoscopy

Chak et al observed that early endoscopy was done in 82% of their patients.

Rockall et al noted that 1108(50.1%) patients underwent early endoscopy which was comparable to that noted in the present study-228(56.2%).

Causes of Upper GI bleed

Barkun et al [11] noted in the RUGBE study, that 56% had peptic ulcer disease as the primary etiology for UGI bleeding, followed by esophagitis (8.4%), Mallory Weiss tears (4.4%) and Dieulafoy lesions (2.5%).

In the present study, 315 patients had non variceal causes of bleed,of which peptic ulcers accounted for 80(25.4%). 52(65%) were duodenal ulcers and 28(35%) were gastric ulcers. Gastritis-174(55.2%)and duodenitis-107(34.8%) accounted for the majority, with oesophagitis seen in 71(22.5%), much higher than that noted by Barkun et al. Mallory Weiss tears accounted for 2(0.6%) cases.

Rockall and Logan studied 2332 cases of UGIB, taking into consideration both variceal and non variceal etiology of bleed. Vreeburg et al.[84] studied 477 subjects presenting with Upper GI bleed in the Amsterdam area. The various causes, documented by them, in comparison to the present study is as follows:

Table 22.Causes of bleed- comparison

	Rockall et al n=2332	Vreeburg etal n=1389	Present study n=406
Peptic ulcer	842(36.1%)	477(34.3%)	80(25.4%)
Malignancy	93(4%)	30(2%)	16(3.9%)
Mallory-Weiss tear	119(5.1%)	56(4%)	2(0.6%)
Oesophagitis	241(10.3%)	155(11%)	71(22.5%)
Gastritis/erosions	240(10.3%)	118(8%)	174(55.2%)
Varices	108(4.6%)	127(9%)	91(22.4%)

A very high percentage of patients(55.2%) were noted to have gastritis, in the present study, as compared to that noted by Rockall et al. and Vreeburg et al. Varices were observed in a much lower percentage by Rockall et al. and Vreeburg (4.6% and 9% respectively), in comparison with this study (22.4%).

In the present study, nonvariceal causes predominated in all age groups , but it was not found to be significantly higher in patients aged >60 years , as compared to those less than 60 years.(P value of 0.11)

Peptic ulcer

Table 23.Distribution of peptic ulcers

Forrest classification	Laine et al[13](%)	Present study(%)
I	18	15
IIA	17	20
IIB	17	18.8
IIC	20	16.3
III	42	30

In the present study, the percentage of ulcers, belonging to various classes of the Forrest classification, were comparable to that noted by Laine et al[13].The percentage of Forrest class III ulcers(30%) were lower than that noted by Laine et al.

Variceal hemorrhage

Sarin et al [85] noted that GOV1 constituted the majority(74%)of gastric varices, followed by GOV2(16%), IGV1(8%) and IGV2(2%). Patients with gastric varices constituted 47(51.6%) of cases in the present study, of which GOV1 accounted for 25.5%, GOV2 formed the bulk with 61.7%, and IGV1 accounted for 12.7%. There were no cases of IGV2.

Endotherapy

Endotherapy was done in 115 cases (28.5%) in the present study. Adrenaline injection was performed in 40 cases (50% of peptic ulcers), Endoscopic sclerotherapy was done in 40 cases and endoscopic variceal ligation in 35 cases (82.4% of variceal bleed).

Vreeburg et al[84]. in their series, noted lower endoscopic intervention rates, with 21% of patients with UGIB in their case series undergoing endotherapy. Injection therapy was instituted in 74% of subjects with bleeding peptic ulcers which was higher when compared to 50% in the present study.

Rockall Score

The mean Rockall score was 4.8 (SD : 1.9, range: 0 -10) in the RUGBE study whereas in the present study, it was lower(mean score : 3.3, S.D:1.7, range: 0-10) indicating that most patients belonged to the low risk category.

Table 24.Risk groups based on Rockall Score

RISK	RUGBE	Rockall et al.	Present study
Low	240(13%)	1058(45.4%)	175(43.1%)
Moderate	999(53%)	1181(50.7%)	178(43.9%)
High	630(34%)	93(3.9%)	53(13%)
Total	1869	2332	406

As compared to the RUGBE study[11] where the majority belonged to the moderate and high risk group, in the present study, most of the patients had a low Rockall score(less than 3) and belonged to the low risk group. However, the percentage of patients belonging to the high risk group, in the present study, was higher than that noted by Rockall et al(13% vs. 3.9%)

Table 25. Comorbidity and hemodyamic instability

	Rockall et al	Present study
Comorbidity	1378(59.1%)	141(34.7%)
Hypotension	256(11.2%)	127(31.2%)

The comorbidities were lower in the present study (34.7%) as compared to that noted by Rockall et al (59.1%),and Yavorski et al(50.9%).[9] but hemodynamic instability was higher (31.2%) than that noted by Rockall et al(11.2%).

Adverse outcome

In the present study, the requirement of blood transfusion was significantly higher in the moderate and high risk groups as compared to the low risk groups, (both P values less than 0.001) , as was the incidence of

rebleed which was more in the moderate and high risk groups as against the low risk group(p<0.001 in both). These findings are compared to that observed by Patel et al.

Table 26.Adverse outcomes-comparison

	Patel et al			Present study		
	Low	Mod	high	low	Mod	high
PRBC reqt	44%	56%	76%	4.6%	40.4%	77.3%
Rebleed	6%	21%	24%	2.3%	15.7%	60.3%

In comparison with the observations of Patel et al, the need for packed red cell transfusions were less in all groups in the present study. But, the percentage of rebleed , was higher than that noted by Patel et al, as far as the high risk groups were concerned.

Akash et al[56], in their study of upper GI bleed, from a tertiary care institution at Chennai,also noted that Rockall score correlated well with the need for blood transfusions, rebleeding and mortality. Of the 100 patients with nonvariceal upper GI bleed studied by them,30 patients had a Rockall score of 1-3 and 28(94%)had no rebleed and did not require transfusions. 28(28%) patients had a clnical score of 4-6, out of which 6(21.4%)had rebleed and 1 patient expired. Of the 20 patients with Rockall score 7-11, 4(20%) patients had rebleed and 7(35%) died and the remaining had prolonged hospital stay. In the present study, the rebleed rate in moderate risk group was 15.6% and slightly lower than that noted by Akash et al.(21.4%) but the rate of rebleed in the high risk group was higher.

The following table gives a comparison of the rebleed noted in the RUGBE[11] study and present study.

Table 27.Risk groups and rebleed- comparison

RISK	RUGBE	Present study
Low	21 (8.8%)	4(2.3%)
Moderate	130(13%)	28(15.7%)
High	107(17%)	32(60.4%)
Total	258	64

The rebleed rates in the present study(15.8%) was comparable to that observed by Barkun et al.[11] viz.13.8% and Rockall et al.(15.4%) but higher than that noted by Yavorski et al(7.1%).

It was noted that in the low risk group, the rebleed was lower in the present study (2.3%) as compared to RUGBE data, where 8.8% rebled and that noted by Rockall et al. [41] where 4.3% had rebleed. But, it was observed that in the high risk group, 60.4% of patients had rebleed which was higher than the rate of 17% noted in the RUGBE data**.**

Limitations of the study

1. As the study centre is a tertiary care institution, referral bias might have occurred, with a higher number of patients with UGIB falling into moderate and high risk groups, which might not be a true reflection of the scenario in the general population.
2. The average length of hospital stay was not documented in the subjects, as was the need for emergency surgery. Thus, the usefulness of Rockall score in validating the above parameters could not be ascertained.

CHAPTER 7

CONCLUSION

- Upper GI bleed is an important indication for endoscopic referral to this institution
- The majority of patients were noted to be less than 60 years of age, with a male predilection.
- Most cases presented with minor or moderate upper GI bleed, with massive bleeds constituting a minority.
- NSAID and alcohol use as well as smoking were notable associated factors.
- Nonvariceal bleed predominated in all age groups, with peptic ulcers and gastroduodenal erosions accounting for the majority of cases.
- Good clinicoendoscopic correlation in varices, MW tears, and duodenal ulcer compared to gastric ulcer and malignancy.
- Based on the Rockall score,most of the cases fell into the low and moderate risk groups(score<6). In the high risk group, the number of patients with variceal bleed were significantly higher compared to the low risk group.
- The rebleed was not significantly higher in patients with varices, as compared to nonvariceal causes.
- The requirement of packed red cell transfusion and rebleed were higher in patients with higher Rockall score (moderate and high risk groups). The mortality was significantly higher in the high risk group as compared to the low risk group. Hence, Rockall score has been found

to correlate well with clinical outcome, including need for transfusions, rebleeding and mortality, and may be used for effective triage of patients into outpatient and inpatient care, as well as to predict prognosis in upper GI bleed.

CHAPTER 8

SUMMARY

Upper GI bleed is one of the most common gastrointestinal emergencies, with considerable morbidity and requires significant hospital resource utilization. The modes of presentation, etiology and clinical spectrum may vary and an interplay of various causative and associated factors have been observed.

Early upper GI endoscopy is of paramount importance in the diagnosis as well as treatment and has also been used to stratify risk in upper GI bleed. Of the various risk stratification scores used, Rockall score is a simple and useful score , based on clinical and endoscopic criteria and has been shown to represent an accurate and valid predictor of rebleeding, mortality and morbidity in the form of blood transfusions and prolonged hospital stay. Thus, it can be used in the triage of patients, to either outpatient care (low risk) or admission and intensive care (moderate or high risk), with anticipation of morbidity and rebleed.

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Plate 1. Forrest Class IIB: Ulcer with adherent clot

Plate 21. Forrest Class IIC: Ulcer with pigmented spot

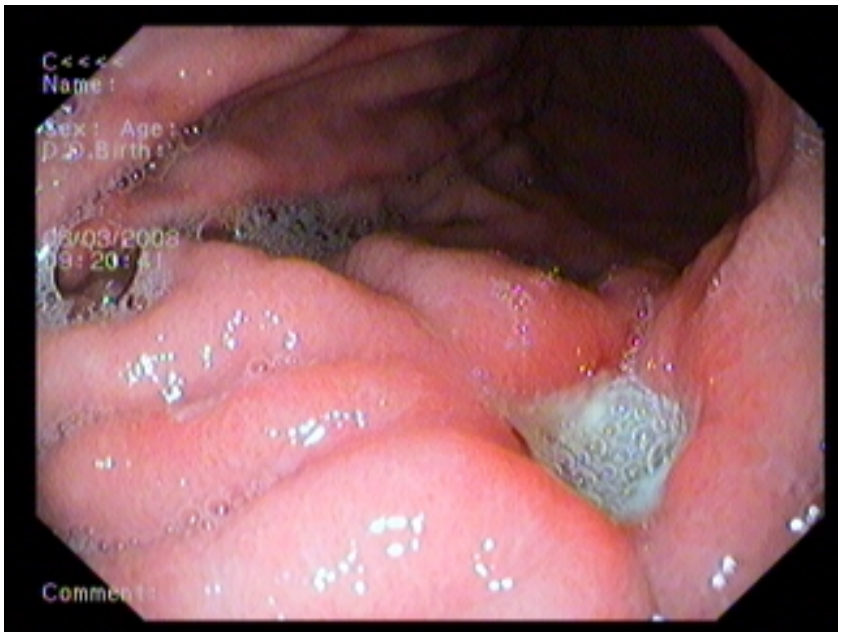

Plate 2 Forrest class III.: ulcer with clean base

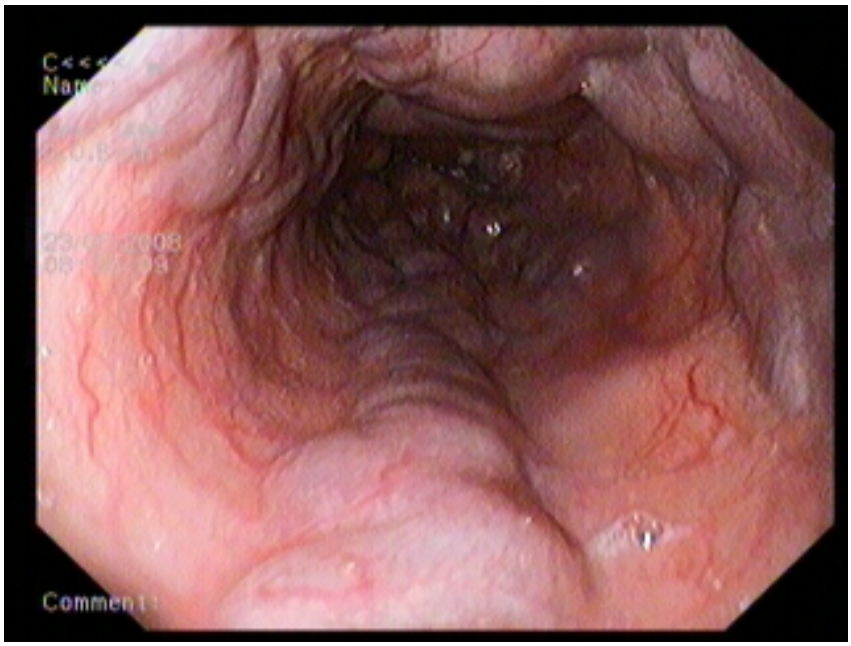

Plate 3. Esophageal varices

APPENDIX

UPPER GASTROINTESTINAL BLEED- CAUSES, ENDOSCOPIC PROFILE AND USEFULNESS OF ROCKALL SCORE

PROFORMA

SL. No

Name

IP Number

GE number

Address

Age

Sex

Date of Admission

Presentation — Hematemesis/Melena/Hematochezia

Severity of bleed — Minor/Moderate/Massive

Associated .factors
- Alcohol
- Smoking
- Corrosive ingestion
- NSAID
- Past GI bleed

Comorbid conditions
- IHD
- CCF
- Renal failure
- Disseminated malignancy

Examination
- Pulse
- Blood pressure
- Pallor
- Abdomen :

Clinical diagnosis

Timing of endoscopy

UGI Endoscopy
- Endoscopic findings
- FORREST class(as applicable)
- SARIN classification
- Grading of varices
- Endoscopic diagnosis

Variceal/nonvariceal

ROCKALL SCORE-COMPONENTS

- AGE
- SHOCK
- COMORBIDITY
- ENDOSCOPIC DIAGNOSIS
- ENDOSCOPIC SRH
- TOTAL Score

RISK GROUP	A/B/C
ENDOTHERAPY	yes/no
Details of endotherapy	Adrenaline/EST/EVL
PRBC transfusion	
Rebleed	Yes/No
Endotherapy for rebleed	Adrenaline/EST/EVL
Death	

S. No	IP. No.	GE	Name	Sex	Presentation	Severity of bleed	Associated factors	Comorbidity	Timing of endoscopy	Endoscopic findings	Forrest Class	Sarin class	Varices-grade	V/NV	Rockall-AGE	Rockall-SHOCK	Rockall-Comorbidity	Endoscopic SRH	DIAGNOSIS	TOTAL Rockall score	Risk group	Blood transfusion	Endotherapy	Rebleed	Endotreapy for rebleed	Death
1	775403	5331/05	Velu	m	H,M	MD	A		E	G,D				NV	0	1	0	0	1	2	A					
2	775414	5333/05	Mani	m	H	MD			E	G,D				NV	0	1	0	0	1	2	A					
3	779361	5330/05	Govindasami	m	H	MN	N, A		E	G,D,GU	1B			NV	0	0	0	0	1	1	A		A	Y	A	
4	779020	5327/05	Poongavanam	m	m	MD	A,S	O	E	V		GOV1	3	V	0	1	2	0	1	4	B	B	EVL			
5	775686	003/06	Mahendra	m	H	MN			E	V		OV	1	V	0	0	0	0	1	1	A					
6	774328	002/06	Shakila	f	H	MN		C	E	G				NV	0	0	2	0	1	3	B					
7	776294	008/06	Dhanapal	m	H,M	MN			E	G,D				NV	0	0	0	0	1	1	A					
8	777554	134/06	Shanthi	f	H,M	MS	S		E	V,PHTG		GOV1	3	V	0	2	2	2	1	7	C	B	EVL	Y	EVL	
9	776788	34/06	Prabhu	m	H	MN			E	G				NV	0	0	0	0	1	1	A					
10	776825	71/06	Mariyam	f	H	MD	A		L	G				NV	0	1	0	0	1	2	A					
11	776281	104/06	Nagalingam	m	H	MD	A,S		L	G,D				NV	0	1	0	0	1	2	A					
12	776627	97/06	Venkatesh	m	M	MD	A,S	L	E	V,PHTG		IGV1		V	0	2	2	2	1	7	C	B				
13	776289	76/06	Chinnakulandai	m	M	MD			L	G,D				NV	1	1	0	0	1	3	B					
14	777346	126/06	Samuel	m	H	MN			E	G,D				NV	0	0	0	0	1	1	A					
15	777372	128/06	Ellammal	f	H	MN			E	O,G,D				NV	1	0	0	0	1	2	A					
16	777206	140/06	Elias	m	H	MN	C		E	O,G				NV	0	0	0	0	1	1	A					
17	777037	138/06	Siva	m	H	MN	A		E	G				NV	0	0	0	0	1	1	A					
18	777538	165/06	Zarina	f	H	MD	B	L	E	V,PHTG,PHTD		GOV1	3	V	0	2	3	2	1	8	C	B	EVL	Y	EVL	D
19	777973	218/06	Nitha	f	H	MN	N,		E	G				NV	1	0	0	0	1	2	A					
20	777983	141/06	Selvaraj	m	H,M	MD	A,S	L	E	V,PHTG,PHTD		GOV1	3	V	0	2	2	0	1	5	B	B	EVL			
21	778535	233/06	Rajamani	m	H,M	MD	A,S	L	E	G,PHTG,PHTD		GOV1	3	V	0	2	2	0	1	5	B	B	EVL			
22	779176	275/06	Jemini	m	M	MD	A	L	L	V,PHTG,PHTD		GOV2	3	V	0	1	2	2	1	6	C	V	EVL	Y	EVL	
23	779827	262/06	Annammal	f	H	MN	A,S		E	MW,O,G				NV	1	0	0	0	1	2	A					
24	779834	268/06	Kumar	m	H	MN	A		L	O,G				NV	0	0	0	0	1	1	A					
25	780317	358/06	Renuka	f	H	MN			E	O				NV	0	0	0	0	1	1	A					
26	780125	345/06	Panneer	m	H,M	MN	A,S		E	O,D				NV	0	0	0	0	1	1	A					
27	780458	369/06	Murugan	m	H	MN	A,S	L	E	V,PHTG,PHTD		GOV2	3	V	0	1	2	0	1	4	B		EVL			
28	780130	360/06	Subramani	m	H,M	MD	A,S	L	E	V,PHTG,PHTD		OV	3	V	0	2	2	2	1	7	C	B	EST	Y	EST	
29	780319	361/06	Muniappan	m	H	MN	A,S		E	O,G				NV	0	0	0	0	1	1	A					
30	779671	304/06	Parthasarathy	m	M	MD	B		E	V		IGV1		V	0	1	0	0	1	2	A					
31	780951	394/06	Jayanti	f	H	MS			E	V		IGV1		V	0	2	0	0	1	3	B	B				
32	781450	208/06	Varadarajan	m	H	MN	N	O	L	G,D,GU	3			NV	0	0	2	0	1	3	B					
33	781758	461/06	Muthalagan	m	H,M	MD	S		E	G,D				NV	0	1	0	0	1	2	A					
34	781836	492/06	Venugopal	m	H	MN	A,S		L	O,GU	2A			NV	1	0	0	2	0	3	B		A			

S. No	IP. No.	GE	Name	Sex	Presentation	Severity of bleed	Associated factors	Comorbidity	Timing of endoscopy	Endoscopic findings	Forrest Class	Sarin class	Varices-grade	V/NV	Rockall-AGE	Rockall-SHOCK	Rockall-Comorbidity	Endoscopic SRH	DIAGNOSIS	TOTAL Rockall score	Risk group	Blood transfusion	Endotherapy	Rebleed	Endotreapy for rebleed	Death
35	781697	464/06	Kamala	f	H	MN	A,S		E	N				NV	0	0	0	0	1	1	A					
36	782075	454/06	Srinivasan	m	H	MN	A		E	G,D				NV	0	0	0	0	1	1	A					
37	781801	461/06	Krishnaveni	f	H	MD		L	E	V,PHTG,PHTD		IGV1		V	0	1	2	0	1	4	B					
38	783063	607/06	Mani	m	H,M	MN			E	D				NV	0	0	0	0	1	1	A					
39	782574	601/06	Ganesan	m	M	MD			E	D				NV	0	1	0	2	1	4	B			Y		
40	783192	599/06	Soundaraj	m	H	MN			E	O,G,D				NV	0	0	0	0	1	1	A					
41	782189	546/06	Anita	f	H,M	MD			E	V		OV	3	V	0	2	0	2	1	5	B		EST			
42	783288	640/06	Suresh	m	H	"MD"	A , S		L	GU	3			NV	0	1	0	0	1	2	A					
43	783214	5240/06	Subramani	m	H,M	MD			L	V,PHTG,PHTD		GOV2	3	V	1	1	0	0	1	3	A	B	EVL			
44	783647	384/06	Munnabhai	m	M	MD		L	E	V,PHTG,PHTD		GOV2	3	V	0	2	3	2	1	8	C	B	EVL			
45	787871	616/06	Balan	m	H,M	MN	A		E	G				NV	0	0	0	0	1	1	A					
46	784113	680/06	Gnanasekar	m	H,bleeding pr	MS	N		E	G				NV	0	2	0	2	1	5	B	B		Y		
47	783706	654/06	Dillibabu	m	h,m	MD	A		E	O,G,D				NV	0	1	0	0	1	2	A	B				
48	784334	662/06	Kuppan	m	h	MN	N	O	E	GU	3			NV	0	0	2	0	1	3	B					
49	784315	678/06	Anjalakshi	f	h	MN		O	E	G				NV	1	0	2	0	1	4	B					
50	785351	727/06	Akila	f	H	MN			E	G				NV	0	0	0	0	1	1	A					
51	784238	792/06	Manivel	m	H,M	MN			L	O,G,D				NV	0	0	0	0	1	1	A					
52	789124	795/06	Dawood	m	M	MN			E	O,G,D				NV	0	0	0	0	1	1	A					
53	785843	693/06	Ramya	f	H	MN	A		E	G				NV	0	0	0	0	0	0	A					
54	785667	872/06	Patchaiappan	m	H	MD	S		L	GJ,V		GOV2	3	V	1	1	2	0	1	5	B	B	EVL			
55	785063	843/06	Manickam	m	H	MN			E	G,MW				NV	0	0	0	0	1	1	A					
56	779208	224/06	Rajendran	m	M	MN			E	G,D	3			NV	0	0	0	0	1	1	A					
57	786453	816/06	Moorty	m	H	MN	A,S		E	N				NV	0	0	0	0	0	0	A					
58	786703	871/06	Kanagavalli	f	H,M	MD		O	E	N				NV	1	1	2	0	0	4	B					
59	785865	801/06	Shanmugam	m	H	MN			E	G				NV	0	0	0	0	1	1	A					
60	787312	942/06	Chandrasekar	m	H	MS	A		L	V,PHTG		IGV1		V	0	2	2	2	1	7	C	B				
61	788204	975/06	Manohar	m	H	MN			E	Varices- grade				NV	0	0	0	0	1	1	A					
62	788712	973/06	Sadayappan	m	H	MN		O	E	O,A				NV	0	0	2	0	1	3	B					
63	787170	423/06	Usman basha	m	H,M	MD			L	O,D				NV	0	1	0	2	1	4	B			Y		
64	789200	1112/06	Jagannathan	m	H,M	MD	A, S		E	G,D				NV	0	1	0	0	1	2	A					
65	787384	945/06	Arumugam	m	H	MD	N	O	E	G				NV	1	1	2	0	1	5	B			Y		
66	789884	1156/06	Saroja	f	H,M	MN	SU		L	D,DU	3			NV	0	0	0	0	1	1	A	B				
67	789888	1186/06	Sivajnanam	m	H	MD	N		E	N				NV	0	0	0	0	0	0	A					
68	790450	1248/06	John Baskar	m	H	MN			E	N				NV	0	0	0	0	0	0	A					

S. No	IP. No.	GE	Name	Sex	Presentation	Severity of bleed	Associated factors	Comorbidity	Timing of endoscopy	Endoscopic findings	Forrest Class	Sarin class	Varices-grade	V/NV	Rockall-AGE	Rockall-SHOCK	Rockall-Comorbidity	Endoscopic SRH	DIAGNOSIS	TOTAL Rockall score	Risk group	Blood transfusion	Endotherapy	Rebleed	Endotreapy for rebleed	Death
69	789019	1194/06	Abdul khader	m	H	MN	A	O	E	G				NV	1	0	2	0	1	4	B					
70	790693	1243/06	Periyasamy	m	H,M	MD			E	O,G,D				NV	0	1	0	0	1	2	A					
71	790989	1248/06	Govindasami	m	H,M	MS		C	L	G,D				NV	1	2	2	0	1	6	C			Y		
72	790421	1216/06	Xavier	m	H,M	MN			E	N				NV	0	0	0	0	0	0	A					
73	789331	1367//06	Shanmugam	m	H,M	MD		L	E	V,PHTG		OV	2	V	0	2	2	0	1	5	B	B	EST			
74	788576	1221/06	Ganesh	m	M	MN		R	L	G				NV	0	0	3	0	1	4	B			Y		
75	791504	1315/06	Napolean	m	H	MN		O	E	N				NV	0	0	2	0	0	2	A					
76	791523	1314/06	Krishnaveni	f	H	MN	S		E	N				NV	0	0	0	0	0	0	A					
77	791482	1312/06	Murali	m	H,M	MD	B	L	L	V,PHTG		OV	2	V	0	2	3	2	1	8	C	B	EST	Y	EST	
78	790946	1251/06	Lakshmiammal	f	M	MN			E	N				NV	1	0		0	0	1	A					
79	790955	1256/06	Alamelu	f	H	MN	N	C	E	N				NV	0	0	2	0	0	2	A					
80	791284	1292/06	Mary	f	H	MN		O	E	G				NV	0	0	2	0	1	3	B			Y		
81	791290	724/06	Nagaraj	m	H	MN	N		E	N				NV	1	0	0	0	0	1	A					
82	792124	1305/04	Kathiresan	m	H	MD		O	E	O				NV	0	1	2	0	1	4	B					
83	792700	1366/06	Krishnan	m	M	MD			L	MS				NV	1	2	0	2	2	7	C					
84	792598	1389/06	Ushar sharif	m	H	MD	B		L	O				NV	2	2	0	2	1	7	C			Y		
85	799092	1390/06	Elumalai	m	M	MD	A,S		E	O,G				NV	0	1	0	2	1	4	B					
86	791827	1765/05	Chakravarti	m	H	MD	A,S		E	V,PHTG		OV	2	V	0	2	2	2	1	7	C	B	EST	Y	EST	
87	792475	1385/06	Durai	m	H	MD	N		E	N				NV	1	1	0	0	0	2	A					
88	794132	1537/06	Rajendran	m	H	MD		L	L	V,		GOV2	3	V	0	2	2	0	1	5	B	B	EVL			
89	794289	1125/06	Viswanathan	m	H,M	MD	N		L	N				NV	1	0	0	0	0	1	A	B				
90	792487	14/06	Muniandi	m	M	MD		O	L	O,MS				NV	0	2	2	2	2	8	C	B				
91	794311	1573/06	Elumalai	m	H,M	MD	A,S	L	E	V,PHTG		OV	2	V	0	2	2	2	1	7	C	B	EST	Y	EST	
92	795036	1551/06	Sundar raman	m	H	MN			E	G				NV	0	0	0	0	1	1	A					
93	792664	1531/06	Samuel	m	H	MN		O.L	L	O,G,D				NV	0	0	2	0	1	3	B			Y		
94	794293	1541/06	Solaiammal	f	M	MD			E	O,G,D,GU	3			NV	0	1	0	0	1	2	A					
95	799583	1581/06	Saroja	f	M	MD		C	L	G				NV	0	1	2	0	1	4	B	B		Y		
96	795626	1574/06	Abhirami	f	H	MN		O	E	N				NV	0	0	2	0	0	2	A					
97	795793	1646/06	Laxmi	f	H,bleeding pr	MD			L	O,D				NV	1	0	0	0	1	2	A					
98	795948	1664/06	Munusamy	m	H	MN	N	O	E	G,D				NV	0	0	2	0	1	3	B			Y		
99	796636	1725/06	Saktivel	m	H,M	MS	A,S		E	O,G,D,DU	3			NV	0	2	0	2	1	5	B	B				
100	796430	1736/06	Nagaraj	m	H	MN			E	G				NV	0	0	0	0	1	1	A					
101	796261	1695/06	Velu	m	bleeding pr	MN			E	N				NV	0	0	0	0	0	0	A					
102	796981	1777/06	Deviraj	m	H,M	MD	N	C,O	E	GU	2B			NV	0	2	2	2	1	7	B	B	A	Y	A	

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103	796635	1724/06	Adilaxmi	m	H	MN	N	O	E	O				NV	1	1	2	0	1	5	B					
104	797208	1686/06	Jeeva	f	H	MN			E	N				NV	0	1	0	0	0	1	A					
105	796672	1776/06	Jayapandian	m	M	MD	A,S		L	,DU	3			NV	0	1	0	0	1	2	A					
106	797313	1806/06	Jagan	m	H,M	MD		L	L	V,PHTG,PHTD		GOV1	2	V	0	1	2	2	1	6	C	B	EVL			
107	797743	1778/06	K+D192artikraja	m	H	MN			L	O,D				NV	0	1	0	0	1	2	A					
108	798313	1868/06	Nayaki	f	M	MD			E	GU	2A			NV	0	1	0	0	1	2	A		A	Y	A	
109	798359	1843/06	Paulsingh	m	H	MN	A,S		L	G,D				NV	0	0	0	0	1	1	A					
110	798238	1838/06	Kasivisalakshi	f	M	MD			E	V,PHTG,PHTD		OV	2	V	0	2	2	0	1	5	B	B	EST			
111	791299	1920/06	Gomati	f	H	MN		R	L	H,D				NV	0	0	3	0	1	4	B					
112	798808	1906/06	Chinnathai	f	H	MN			L	N				NV	0	0	0	0	0	0	A					
113	799225	1904/06	Venkatesan	m	M	MD	N		E	G,GOO				NV	1	1	0	0	1	3	B					
114	799118	1962/06	Kannan	m	H	MD			L	V,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EVL			
115	798628	1378/06	Kalpana	f	H	MN	B	L	E	V		OV	1	V	0	2	0	2	1	5	B	B				
116	799496	1916/06	Selvi	f	H	MN			E	N				NV	0	0	0	0	0	0	A					
117	799401	1014/06	Vijayan	m	H	MN	S		E	N				NV	0	0	0	0	0	0	A					
118	799902	1897/06	Madarasi	m	H	MN	S		L	G,DU	2A			NV	0	0	0	0	1	1	A		A	Y	A	
119	799742	1992/06	Santosh	m	H	MD			L	N				NV	0	1	0	2	1	4	B					
120	799418	3824/05	Jayalakshmi	f	M	MD			E	V		OV	2	V	0	2	2	0	1	5	B	B	EST	Y	EST	
121	800778	2102/06	Sukanya	f	H	MN			E	O,G,D				NV	0	0	0	0	1	1	A					
122	800387	2106/06	Saravanan	m	H,M	MD	S		L	G,D,GU	2A			NV	0	1	0	0	1	2	A		A	Y	A	
123	769842	2069/06	Manimegalai	f	H,M	MD			E	O				NV	0	1	0	0	1	2	A					
124	800979	2003/06	Palani	m	H,M	MD	A,S		E	O,D				NV	0	1	0	0	1	2	A					
125	801411	2133/06	Saraswati	f	H	MN			E	D				NV	0	0	0	0	1	1	A					
126	801558	2154/06	Ethiraj	m	H,M	MD	A,N,S	C,L	L	G,D,GU	2A			NV	0	1	2	0	1	4	B	B		Y	A	
127	801234	1121/O6	Viswan	m	H,M	MD	N		L	N				NV	1	1	0	0	0	2	A					
128	793487	1411/06	Muniappan	m	M	MD			L	MS				NV	0	1	0	2	2	5	B					
129	794321	1572/06	Sivan	m	H,M	MD		L	E	V,PHTG		OV	3	V	0	2	2	0	1	5	B	B	EST			
130	795038	1552/06	Sundar rajan	m	H	MN			L	G				NV	0	0	0	0	1	1	A					
131	792663	1533/06	Shyam	m	H	MN		L,O	L	O,G,D				NV	0	0	2	2	1	5	B					
132	794298	1543/06	Saraswathi	f	M	MD			E	GU	2A			NV	0	2	0	0	1	3	B		A			
133	794294	1581/06	Sarojini	f	M	MD		C	L	G,GU	2A			NV	0	2	2	0	1	5	B	B	A			
134	795627	578+C170/(	Archana	f	H	MN		O	E	N				NV	0	0	2	0	0	2	A					
135	795793	1648/06	Laxmi	f	H,bleeding pr	MD			L	O,D				NV	1	1	0	0	1	3	B	B				
136	795347	1665/06	Munusamy	m	H	MN	N	O	E	G,D				NV	0	0	2	0	1	3	B			Y		

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137	796637	1727/06	Sakti	m	H,M	MS	A,S	O	E	G,D,DU	2A			NV	0	2	2	0	1	5	B					
138	796432	1738/06	Nagaraj	m	H	MN			E	G				NV	0	1	0	0	1	2	A					
139	796263	1696/06	Velayudhan	m	bleeding pr	MN			E	N				NV	0	0	0	0	0	0	A					
140	796988	1778/06	Devaraj	m	H,M	MD	N,S	C,O	E	GU	2B			NV	0	2	2	2	1	7	C	B	A	Y	A	
141	796645	1728/06	Gajalaxmi	f	H	MN	N	O	E	O				NV	1	1	2	2	1	7	C			Y		
142	797255	1634/06	Jeeva	f	H	MN			E	N				NV	0	0	0	0	0	0	A					
143	796678	1778/06	Pandian	m	M	MD	A,S		L	O,G,D,GU	2A			NV	0	0	0	0	1	1	A		A			
144	797318	1886/06	Jagannivas	m	H,M	MD		L	L	V,PHTG,PHTD		GOV1	3	V	0	2	3	2	1	8	C	B	EVL	Y	EVL	D
145	797704	1787/06	Kartik	m	H	MN			L	O,D				NV	0	0	0	0	1	1	A					
146	798313	1888/06	Periyanayaki	f	M	MD			E	GU	2A			NV	0	0	0	2	1	3	B		A	Y	A	
147	798368	1848/06	Paul	m	H	MN	A,S		L	G,D				NV	0	0	0	0	1	1	A					
148	798239	1848/06	Visalakshi	f	M	MD			E	V,PHTG,PHTD		GOV2	3	V	0	2	2	0	1	5	B	B	EST			
149	791300	1902/06	Srimati	f	H	MN		R	L	H,D				NV	0	0	3	0	1	4	B					
150	798808	1908/06	Chinnammal	f	H	MN			L	N				NV	0	0	0	0	0	0	A					
151	799240	1900/06	Venkateswaran	m	M	MD	N		E	G,GOO				NV	1	1	0	0	1	3	B					
152	799218	1966/06	Kamalakannan	m	H	MD			L	,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EST			
153	798630	1307/06	Krishnan	m	H	MN	B	L	L	V		OV	2	V	0	0	0	2	1	3	B	B	EST			
154	799497	1916/06	Selvam	f	H	MN			E	N				NV	0	1	0	0	0	1	A					
155	799400	1041/06	Vijayakumar	m	H	MN	S		E	N				NV	0	0	0	0	0	0	A					
156	799920	1145/06	Madavan	m	H	MN			E	G				NV	0	1	0	0	1	2	A					
157	799742	1998/06	Santosh	m	H	MD			L	N				NV	0	1	0	0	1	2	A					
158	799428	3824/05	Jaya	f	M	MD	A	L	L	V		OV	2	V	0	1	2	0	1	4	B	B	EST			
159	800778	2103/06	Kalaiselvan	f	H	MN			E	O,G,D				NV	0	0	0	0	1	1	A					
160	800386	2160/06	Muthuvel	m	H,M	MD	A		L	G,D				NV	0	2	0	0	1	3	B			Y		
161	769844	2070/06	Manimalai	f	H,M	MD			E	H,O				NV	0	1	0	0	1	2	A					
162	800976	2007/06	Velu	m	H,M	MD	A		L	O,D				NV	0	1	0	0	1	2	A					
163	801419	2144/06	Sumati	f	H	MN			E	D				NV	0	0	0	0	1	1	A					
164	801555	2155/06	Govindaraj	m	H,M	MD	A,N	C,O	L	G,D				NV	0	1	2	0	1	4	B			Y		
165	811775	4325/04	Jeevarathinam	m	M	MD			E	V		GOV2	3	V	0	2	3	2	1	8	C	B	EST	Y	EST	
166	812824	3002/06	Gopu	m	H,M	MD			E	O,G				NV	0	2	0	0	1	3	B					
167	812136	2997/06	Sheikh meeran	m	H,M	MD		L	L	V,PHTG		GOV2	3	V	1	2	2	2	1	8	C	B	EST	Y		
168	786670	2734/06	Shankaran	m	H	MN	N		E	G,DU	2A			NV	1	1	0	0	1	3	B		A			
169	812576	2961/06	Subaida	f	H	MN	S		L	G				NV	0	1	0	0	1	2	A					
170	812050	2956/06	Laxmi	f	H	MN	A	L	L	V,PHTG		OV	2	V	0	2	2	2	1	7	C		EST		EST	

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171	812344	2922/06	Arumugam	m	H,M	MS	B,N		E	O				NV	0	2	0	2	2	6	C			Y		
172	814115	2982/06	Raja	m	H	MN	B,A		L	O,G,D				NV	0	0	0	0	1	1	A					
173	813273	3013/06	Shanmugam	m	H	MN	A,N		E	G				NV	1	1	0	0	1	3	B			Y		
174	814911	3022/06	Ramani	f	H	MN	N		L	V		OV	2	V	0	1	2	0	1	4	B		EST			
175	815664	3195/06	Shankar	m	H,M	MD	B,S	L	L	GU	2A			NV	0	2	2	2	1	7	C	B	A	Y	A	
176	816452	4451/06	Abdul musharaf	m	H,M	MS			L	V,PHTG		GOV2	3	V	0	2	3	0	1	6	C	B	EVL	Y	EVL	
177	818088	3390/06	Natarajan	m	H,M	MD			E	G,D				NV	1	2	0	0	1	4	B			Y		
178	816832	3337/06	Kalipillai	m	M	MD	N,S		L	DU	1B			NV	1	1	0	2	1	5	B		A	Y	A	
179	818377	3385/06	Devika	f	H	MN			L	N				NV	0	0	0	0	0	0	A					
180	818375	3260/06	Naveen	m	H	MD		L	L	MS				NV	0	2	0	2	2	6	C	B				
181	15083	3399/06	Rekha	f	H,M	MD			E	N				NV	0	0	0	0	0	0	A					
182	812928	2272/06	Paryal	f	H,	MN	B,N		L	GU	2A			NV	0	0	0	0	1	1	A		A			
183	818934	3373/06	Manonmani	f	H	MN			E	N				NV	0	0	0	0	0	0	A					
184	819158	3455/06	Govindaraj	m	H	MN	N		L	P				NV	1	0	0	0	1	2	A					
185	819473	1235/06	Gopinath	m	H	MN	C		E	#NAME?				NV	0	0	0	0	1	1	A					
186	819483	3353/06	Ravi	m	H	MD	A		L	O				NV	0	1	0	0	1	2	A					
187	819924	3519/06	Subhash	m	H,M	MD	A		L	G				NV	0	1	0	0	1	2	A					
188	818085	3369/06	Abdul Jaffer	m	H	MD		L	E	V,PHTG,PHTD		OV	1	V	0	1	2	2	1	6	C	B				
189	819415	3484/06	Sukumar	m	H,M	MD			L	O,G,D				NV	0	1	0	0	1	2	A					
190	819924	3519/06	Subhash	m	H,M	MD	A,S		L	DU	1B			NV	0	1	0	2	1	4	B		A			
191	820269	3563/06	Laxmi	f	H<M	MD			L	V,PHTG		OV	2	V	0	2	2	0	1	5	B	B	EST			
192	820650	3456/06	Saravanan	m	H,M	MN	B	L	L	N				NV	0	0	2	0	0	2	A					
193	819406	3483/06	Rajamani	m	H,M	MD	A	L	L	V,PHTG		OV	2	V	0	2	2	0	1	5	B		EST			
194	820624	2245/06	Dhanalaxmi	f	H,M	MD	S		L	V,PHTG		GOV2	3	V	0	2	0	0	1	3	B	B	EVL			
195	818941	3642/06	Sivalingam	m	H	MN		O	L	O				NV	1	0	2	0	1	4	B					
196	821572	3661/06	Bhaskaran	m	H	MN	N	C,O	E	G				NV	0	0	2	0	1	3	B					
197	822073	3668/06	Srinivasan	m	H	MN			E	G,D				NV	0	0	0	0	1	1	A					
198	821863	3685/06	Kaliyaperumal	m	H	MN	S		E	O,G,D				NV	0	0	0	0	1	1	A					
199	821128	3626/06	Annamalai	m	H	MN	A		E	O,G,D				NV	1	0	0	0	1	2	A					
200	822870	2501/05	Kuppan	m	H	MN			L	O,GOO				NV	0	0	0	0	1	1	A					
201	823075	3769/06	Selvaraj	m	H	MN			E	G				NV	1	1	0	0	1	3	B					
202	823150	3761/06	Kasinath	m	H	MD	A		L	G				NV	0	1	0	0	1	2	A					
203	823798	3796/06	Veeramuthu	m	H,M	MD	N		E	G,D				NV	0	1	0	0	1	2	A					
204	823261	3804/06	Chakravarthi	m	H,M	MD		L	L	V,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EVL			

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205	823082	3801/06	Natesan	m	H,m	MS	N,S		L	DU	3			NV	1	2	0	2	1	6	C	B				
206	822259	3770/06	Manohar	m	M	MD			L	G				NV	0	2	0	0	1	3	B					
207	822597	3777/06	Mohammed	m	H,M	MD	N,S		E	G,D,DU	3			NV	0	1	0	0	1	2	A					
208	821108	3762/06	Murugan	m	H	MN			E	O,G				NV	0	0	0	0	1	1	A					
209	823929	3821/06	Usman Ali	m	H,M	MD			L	G				NV	0	1	0	0	1	2	A					
210	827382	3801/06	Natesan	m	H,M	MD	N		E	G,D				NV	1	2	0	0	1	4	B			Y		
211	820555	3821/06	Vasanthi	f	M	MD		O	L	DU	2B			NV	0	2	2	2	1	7	C	B	A	Y	A	
212	824091	3766/06	Ramachandran	m	H	MD			E	V,PHTG		OV	2	V	0	2	2	0	1	5	B		EST			
213	824494	3884/06	Karunanidhi	m	H,	MN		L	E	V,PHTG		GOV1	3	V	0	2	2	2	1	7	C	B	EVL	Y	EVL	
214	824672	3814/06	Sundaram	m	H	MN	A		L	G				NV	0	0	0	0	1	1	A					
215	824661	3882/06	Ramesh	m	H, M	MD	S	L	L	DU	2A			NV	0	2	0	2	1	5	B	B	A			
216	824666	3801/06	Anjana	f	M	MD			L	G				NV	0	1	0	0	1	2	A					
217	825036	2955/06	Rasheed	m	H	MN	S	O	E	G,D,GU	3			NV	1	1	2	0	1	5	B					
218	825097	3319/06	Selvan	m	H,M	MD		L	L	V,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EVL			
219	825214	3762/06	Murugan	m	H	MN			E	V,PHTG		GOV1	3	V	0	1	2	0	1	4	B	B	EVL			
220	825295	3914/06	Subramani	m	M	MN			E	G				NV	0	0	0	0	1	1	A					
221	825502	4037/06	Muthaiah	m	m	MD	S	O	L	G,DU	3			NV	1	1	2	0	1	5	B					
222	825908	4054/06	Pennciliah	m	M	MD	A	L	L	V,PHTG		GOV2	3	V	0	2	2	2	1	7	C	B	EVL			
223	824948	3942/06	Anjalai	f	H, M	MD		O	L	G				NV	1	1	2	0	1	5	B					
224	826409	3896/06	Rani	f	H	MN			E	N				NV	0	1	0	0	0	1	A					
225	826382	3680/02	Ilangovan	m	H,m	MD	B	L	L	V,PHTG		OV	3	V	0	2	3	2	1	8	C	B	EST	Y	EST	D
226	826621	4098/06	Rajendran	m	H	MN	A	L	L	V,PHTG		OV	2	V	0	1	2	0	1	4	B		EST			
227	827062	4021/06	Gomati	f	M	MD		C	E	G				NV	0	2	2	0	1	5	B					
228	826910	4122/06	Babu	m	H	MN			L	G,D				NV	0	0	0	0	1	1	A					
229	826095	4119/06	Ganesan	m	H	MD			L	O,G,D				NV	2	2	0	0	1	5	B					
230	827378	4138/06	Palani	m	M	MD	B,S		L	O,G,DU	1B			NV	0	2	0	2	1	5	B	B	A			
231	827533	3455/06	Gowndan	m	H,M	MD			L	MS				NV	1	2	0	2	2	7	C			Y		
232	827765	1570/06	Abhirami	f	H,	MD			E	V		OV	3	V	0	2	0	2	1	5	B	B	EST			
233	825913	3924/05	Narayana moorty	m	H	MD	B,SU	L	L	V,PHTG		GOV1	3	V	0	2	2	0	1	5	B	B	EVL			
234	827840	4229/06	Ganesan	m	H,M	MD	A	L	L	MS				NV	0	2	2	2	2	8	C	B		Y		
235	828498	4201/06	Krishnan	m	H, M	MS	SU		L	GJ,G				NV	0	2	0	0	1	3	B					
236	829804	4271/06	Jeevita	f	H	MN			E	G				NV	0	0	0	0	1	1	A					
237	824655	4225/06	Kumar	m	M	MD	A,S		L	G,D,GU	2C			NV	0	2	0	0	1	3	B	B				
238	829748	4262/06	Annammal	f	H	MN			L	O,G,D				NV	1	0	0	0	1	2	A					

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239	829392	4325/06	Mannankatti	m	H	MN	B,C		E	G				NV	0	1	0	0	1	2	A					
240	829717	4330/06	Pandurangan	m	H	MD	B,S		L	G,D,GU	2C			NV	0	1	0	0	1	2	A					
241	829442	3824/06	Jayalakshmi	f	H,M	MS	A,B	L	E	V		OV	3	V	0	2	2	2	1	7	C	B		Y	EVL	
242	828854	4269/06	Saravanan	m	H	MN			L	O,G,D				NV	0	0	0	0	1	1	A					
243	830334	2662/05	Krishnaveni	f	H	MN	B		E	O,G,D				NV	0	0		0	1	1	A					
244	830006	3668/06	Srinivasan	m	H	MD	B		L	DU,GOO	2C			NV	0	1		0	1	2	A					
245	830332	4228/06	Kumaran	m	H	MN	S	C	E	G,D				NV	0	0	2	0	1	3	B			Y		
246	825913	3928/05	Narayanan	m	H	MD			L	V,PHTG		GOV1	3	V	0	1	2	2	1	6	C	B	EVL			
247	828922	3909/06	Sittrarasan	m	H	MN	B,A		L	V		GOV2	3	V	0	1	2	0	1	4	B	B	EVL			
248	830571	4375/06	Sampath	m	M	MD	A,S		E	G,DU	2B			NV	0	2	0	2	1	5	B		A			
249	830682	4373/06	Papasami	m	H,M	MD	S		L	GU	2C			NV	1	2	0	0	1	4	B					
250	830820	4331//06	Saktivel	m	H	MN	N		E	G,D				NV	0	0	0	0	1	1	A					
251	830877	4370/06	Gunasekar	m	H	MD	N		L	G				NV	0	1	0	0	1	2	A					
252	830819	2811/05	Palani	m	H	MD	N	L	L	V,PHTG		OV	2	V	0	2	2	0	1	5	B	B	EST			
253	831178	4417/06	Dakshinamoorthy	m	H,M	MD	N		E	G,D				NV	0	1	0	0	1	2	A					
254	831180	1648/06	Sundar	m	H,M	MS	B		E	V,PHTG		OV	2	V	0	2	0	2	1	5	B					
255	830900	1656/06	Subadhra	f	M	MD		O	E	G				NV	0	2	2	0	1	5	B					
256	831358	439/06	Ethiraj	m	H	MN	S		E	G,DU	2C			NV	1	0	0	0	1	2	A					
257	830921	4419/06	Nathan	m	H,M	MS		O	E	DU	2B			NV	0	2	2	2	1	7	C	B	A	Y	A	
258	831550	4478/06	Visalakshi	f	H,M	MD	N		L	G,D,GU	2C			NV	0	2	0	0	1	3	B					
259	830780	4380/06	Chinnaponnu	f	H, M	MS		L	L	V,PHTG		GOV2	3	V	0	2	3	0	1	6	C	B	EVL	Y	EVL	
260	831797	4456/06	Saroja	f	H, M	MN	N		E	MS				NV	0	0	0	2	2	4	B					
261	831983	4315/06	Devaki	f	H	MN	A,S		E	G				NV	0	0		0	1	1	A					
262	831882	4454/06	Lourdusami	m	H,M	MD	SU		L	O,G,D				NV	0	1		0	1	2	A					
263	831443	4434/06	Munusamy	m	H,M	MD			L	A				NV	0	1		2	1	4	B	B				
264	831089	4394/06	Vijayan	m	M	MD		L	E	V				V	0	2	2	0	1	5	B	B				
265	831812	4490/06	Manimozhi	f	H	MD		L	L	V,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EST			
266	832582	4414/06	Stella	f	H	MN	N		L	G				NV	0	1	0	0	1	2	A					
267	832587	4406/05	Ravi	m	H	MN	A,S	L	E	G				NV	0	1	2	0	1	4	B					
268	832485	4498/06	Panjalai	f	H	MN		O	L	O,G,D				NV	0	0	2	0	1	3	B					
269	830058	4380/06	Amir basha	m	M	MD	A	L	E	V		OV	2	V	0	2	2	0	1	5	B	B	EST			
270	831172	4420/06	Chinnaiya	m	H	MD	N,S	R	L	DU	2B			NV	2	2	3	2	1	10	C		A	Y	A	
271	833039	4538/06	Kamaludeen	m	H	MN	A,S	L	E	V		OV	2	V	1	2	2	2	2	9	C	B		Y	EST	
272	833403	4545/06	Dayalan	m	H,M	MD	A,S	L	E	V,PHTG		OV	2	V	0	2	2	0	1	3	B	B	EST			

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273	833734	4323/06	Maharun beevi	f	H	MD		L	L	V,PHTG		GOV2	3	V	0	2	3	2	1	8	C	B	EVL			
274	833197	4625/06	Selvi	f	H	MN	N	O	E	H,O,D				NV	0	0	2	0	1	3	B					
275	834056	4616/06	Vasu	m	H	MN		C	L	O,G,D				NV	0	0	2	0	1	3	B					
276	834163	4166/06	Dhanalaxmi	f	H	MN			E	G				NV	1	1	0	0	1	3	B					
277	834110	4623/06	Deepa	f	H	MN	C		E	G				NV	0	0	0	0	1	1	A					
278	832200	4620/06	Kanniappan	m	H	MN	S		L	DU	2A			NV	0	0	0	2	1	3	B		A			
279	834699	4464/06	Mahalaxmi	f	M	MD			L	DU	2B			NV	0	1	0	2	1	4	B		A			
280	834700	4580/06	Siraj	m	H,M	MD	N		E	N				NV	0	1	0	0	0	1	A					
281	834721	4639/06	Murugan	m	H	MN			L	V		OV	2	V	0	1	2	0	1	4	B	B	EST			
282	835298	4604/06	Baby	f	H	MN		C	L	G,D				NV	0	0	2	0	1	3	B					
283	835283	4723/06	Vasanta	f	H	MN	N		E	G,D				NV	0	0	0	0	1	1	A					
284	834744	4706/06	Kasiammal	f	H	MN		O	L	G,D				NV	0	0	2	0	1	3	B					
285	835193	4730/06	Vijayalaxmi	f	H	MN	A,N		L	W				NV	0	0	0	0	1	1	A					
286	835186	4691/06	Srinivasan	m	H	MD	N,S		L	G,D,GU	2C			NV	0	1	0	0	1	2	A					
287	835201	4692/06	Rajkumar	m	H,M	MS			L	V,PHTG		GOV2	3	V	0	2	0	0	1	3	B	B	EVL			
288	835881	46826/06	Murugesan	m	H	MN	A,S		E	G,D				NV	0	0	0	0	1	1	A					
289	835883	4595/06	Kumari	f	H	MD	N		L	N				NV	0	1	0	0	0	1	A					
290	836176	4752/06	Raman	m	H,M	MD	S		E	BD,DU	3			NV	1	1	0	0	1	3	B	B				
291	834484	4655/06	Soundararajan	m	H,M	MD	N		E	V,PHTG,PHTD		GOV2	3	V	0	2	2	2	1	7	C	B	EST	Y		
292	836382	4767/06	Masila	m	H	MD			L	DU	2C			NV	0	2	0	0	1	3	B					
293	836786	4813/06	Murugan	m	H	MN	A,S		E	G				NV	0	0	0	0	1	1	A					
294	836988	4484/06	Sahadevan	m	H	MN			E	G,D				NV	0	0	0	0	1	1	A					
295	837537	4852/06	Ramesh	m	H,M	MD	N,S		L	DU	1B			NV	0	2	0	2	1	5	B	B	A			
296	837540	4851/06	Sindhuja	f	M	MN	N		E	G,D				NV	0	1	0	0	1	2	A					
297	837592	4857/06	Senthil kmar	m	H,M	MD	N,S	L	L	G,DU	2A			NV	0	2	2	2	1	7	C		A	Y	A	
298	837567	4858/06	Kuppusamy	m	H	MS	A,S	L	E	V,PHTG		OV	2	V	0	2	2	2	1	7	C	B		Y	EST	
299	837260	4836/06	Muniammal	f	H,M	MD	N		L	G				NV	0	1	0	0	1	2	A					
300	838203	4721/06	Loganayaki	f	H,M	MD			L	G,DU	2B			NV	0	1	0	2	1	4	B	B	A	Y	A	
301	838054	4922/06	Gopal	m	H	MD	S		L	GJ,G,D				NV	0	2	0	0	1	3	B					
302	838357	4932/06	Kalaiselvan	m	H	MD	N		E	DU,GOO	2B			NV	0	2	0	2	1	5	B	B		Y	A	
303	838785	4919/06	Luminachandra	f	H	MN			E	G				NV	0	0	0	0	1	1	A					
304	838786	4481/06	Bavani	f	H,M	MD			E	G				NV	0	1	0	0	1	2	A					
305	839432	4881/06	Gnanamary	f	H	MN			E	G,D				NV	0	0	0	0	1	1	A					
306	838781	4970/06	Muthu	m	H	MN	N,A,S		L	DU	2B			NV	0	0	0	2	1	3	B		A			

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307	839443	4887/06	Balu	m	H	MD	N,S		E	GU	2C			NV	0	2	0	0	1	3	B					
308	839441	4937/06	Menaka	f	H	MD	N		L	G,D				NV	0	2	0	0	1	3	B					
309	838359	4944/06	Kannabiran	m	H	MD			L	G,D				NV	1	1	0	0	1	3	B					
310	839261	4960/06	Selvakumar	m	M	MD			L	G,D				NV	0	1	0	0	1	2	A	B				
311	840236	5063/06	Alagesan	m	H	MN	A		E	G,GU	3			NV	0	0	0	0	1	1	A					
312	839143	4993/06	Anjamma	f	H	MS			L	G,D				NV	0	2	0	2	1	5	B					
313	840280	5064/06	Sakunthala	f	H	MD			L	DU	2B			NV	0	1	0	2	1	4	B	B	A			
314	839862	5109/06	Ramesh	m	H,M	MD	A		E	G				NV	0	2	0	0	1	3	B					
315	840390	5131/06	Raja	m	H,M	MN			L	G				NV	0	0	0	0	1	1	A					
316	840234	5096/06	Sukumar	m	H	MD	A	L	L	G				NV	0	1	2	0	1	4	B					
317	839120	3319/06	Selvi	f	H	MN			E	G				NV	0	0	0	0	1	1	A	B				
318	840964	5085/06	Vennila	f	H	MD			L	GJ,GU	2C			NV	0	2	0	2	1	5	B	B				
319	840869	5088/06	Varadan	m	H	MN	A		L	V,PHTG		GOV2	3	V	1	1	2	2	1	7	C	B	EVL		EVL	
320	841044	5138/06	Sekar	m	H	MN	A		E	V,PHTG		GOV2	3	V	0	1	2	0	1	4	B	B	EVL			
321	841566	5182/06	Beena mary	f	H	MN	N	L	E	V,PHTG		GOV2	3	V	0	2	2	2	1	7	C		EST		EST	
322	841454	5146/06	Ismail	m	H, bleeding PR	MD	A,S	L	E	V,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EVL			
323	841873	5204/06	Ramesh	m	H	MN		L	L	V,PHTG		OV	2	V	0	1	2	0	1	4	B	B	EVL			
324	842635	4336/05	Rajendran	m	H,M	MD		L	E	V,PHTG		OV	2	V	0	2	2	2	1	7	C		EST		EST	
325	842673	4422/06	Rajesh kumar	m	H	MN	A		E	V,PHTG		OV	2	V	0	2	0	0	1	3	B		EST			
326	842714	5217/06	Krishnan	m	H	MD	SU	L	L	V,PHTG		OV	3	V	0	2	2	0	1	5	B			Y	EVL	
327	842615	5257/06	Selvaraj	m	H	MN	C		E	V,PHTG		OV	1	V	0	1	0	0	1	2	A					
328	840446	5120/06	Gnanasekaran	m	M	MD		L	L	V,PHTG		GOV2	3	V	0	1	2	2	1	6	C	B	EST			
329	843630	5323/06	Moorty	m	H,M	MD	B	L	E	V,PHTG		GOV2	3	V	0	2	2	0	1	5	B	B	EST			
330	842999	5267/06	Ponnusamy	m	H	MN	A	L	L	V,PHTG		GOV2	3	V	0	1	2	0	1	4	B		EST			
331	843646	5321/06	Banumathy	f	H,M	MD			L	V,PHTG		OV	2	V	0	2	2	0	1	5	B	B	EST			
332	843519	5332/06	Guhan	m	H	MN	A,S		E	V,PHTG		OV	2	V	0	1	0	0	1	2	A					
333	844265	5367/06	Mookayee	f	H	MD		L	L	V,PHTG		IGV1		V	1	2	2	0	1	6	C	B				
334	844308	5371/06	Lakshmi	f	H,M	MD	N		E	V,PHTG		OV	2	V	0	2	0	2	1	5	B	B	EST		EST	
335	844808	5241/06	Dhanalaxmi	f	H	MN			L	V,PHTG		OV	2	V	0	1	0	0	1	2	A					
336	844028	5339/06	Jothi	f	H	MN	A	L	E	V,PHTG		OV	2	V	0	1	2	0	1	4	B		EST			
337	844345	5385/06	Viswanathan	m	H	MN	N	C	E	V,PHTG		OV	1	V	0	1	2	0	1	4	B					
338	844748	5409/06	Kalidas	m	H	MN	N		E	V,PHTG		GOV1	3	V	0	1	0	0	1	2	A	B	EVL			
339	844744	5442/06	Guganraj	m	H	MN	N		L	V,PHTG		OV	1	V	0	2	0	0	1	3	B	B				
340	845009	5462/06	Dayalan	m	H	MD	A	L	E	V,PHTG		OV	1	V	0	1	2	0	1	4	B	B				

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341	845895	5074/06	Minnala	f	H	MN			L	V,PHTG		OV	1	V	1	1	2	0	1	5	B	B				
342	846210	5512/06	Rajkumar	m	H	MD	C		E	O,G,D				NV	0	1	0	0	1	2	A					
343	846156	5511/06	Indira	f	M	MD	A		L	N				NV	0	0	0	0	0	0	A					
344	846135	5510/06	Gowri	f	M	MD	A		L	N				NV	0	0	0	0	0	0	A					
345	846668	5517/06	Rathinam	m	H,M	MD	N		E	GU	3			NV	1	2	0	0	1	4	B					
346	846760	5515/06	Chinnaponnu	f	H	MN	N	C	E	G,D				NV	1	1	2	0	1	5	B	B				
347	846612	5518/06	Kodeeswaran	m	H,M	MD	B		E	G				NV	0	1	0	0	1	2	A					
348	847008	5533/06	Subramani	m	H	MN	A		E	G				NV	0	0	0	0	1	1	A					
349	845102	5548/06	K+D87rishnaraj	m	H	MN	N		L	O,D,GU	3			NV	0	0	0	0	1	1	A					
350	847554	5554/06	Muthuraman	m	H,M	MD	N,S		E	O,D,DU	3			NV	0	1	0	0	1	2	A					
351	847584	5578/06	Kanniammal	f	H,M	MD			L	G,D				NV	1	1	0	0	1	3	B					
352	847997	5588/06	Natarajan	m	H,M	MD	N		E	G				NV	0	2	0	0	1	3	B					
353	848103	5613/06	Ramamoorthy	m	H,M	MN	N		E	O,G				NV	1	1	0	0	1	3	B					
354	896106	5617/06	Dhanasekhar	m	H,M	MD	N,S	C	E	G,D,DU	3			NV	0	1	2	0	1	4	B					
355	848300	5658/06	Venugopal	m	H,M	MD		L	L	G				NV	0	1	2	0	1	4	B					
356	849439	5666/06	Mahalaxmi	f	H	MN			L	,DU	3			NV	0	1	0	0	1	2	A					
357	849437	5564/06	Vinayagam	m	H	MN	A		E	G				NV	0	0	0	0	0	0	A					
358	849674	5709/06	Jamesh babu	m	H	MN			E	G+L203				NV	0	0	0	0	0	0	A					
359	849322	5736/06	Vasanta	f	H	MN	N	C.O	E	MS				NV	1	0	2	2	1	6	B					
360	849344	5703/06	Loganathan	m	H	MN			E	MS				NV	0	1	0	2	2	5	B	B				
361	850228	5777/06	Roshini	f	H	MN			E	H,G,D				NV	0	1	0	0	1	2	A					
362	850925	5839/06	Selvaraj	m	H	MN	N		L	MS				NV	0	1	0	2	2	5	B					
363	850806	5837/06	Kallel	m	M	MD	A,N	O	L	MS				NV	0	2	2	2	2	8	C	B				
364	850819	5790/06	Govindaraj	m	H	MD	S		E	MS				NV	0	2	0	2	2	6	C	B				
365	850748	5845/06	Saraswati	f	H	MN			E	MS				NV	0	2	0	2	2	6	C	B				
366	852197	5917/06	Prakash	m	H,M	MD	C		E	G,D				NV	0	2	0	0	1	3	B	B				
367	850761	5858/06	Subramani	m	H,M	MD			E	O,G,D				NV	0	2	0	2	1	5	B					
368	852046	5943/06	Kandasamy	m	M	MD			E	G				NV	1	1	0	0	1	3	B					
369	852831	5761/06	Malini	f	H	MN			L	GU	2B			NV	0	2	0	2	1	5	B	B				
370	853196	1197/06	Deepa	f	H,M	MD	A	L	L	V,DU	3			NV	0	2	2	0	1	5	B	B				
371	852478	3202/06	Kavita	f	H,M	MD			E	G,DU	3			NV	0	1	0	0	1	2	A					
372	853791	6060/06	Govindan	m	H,M	MD	A		L	G,D				NV	0	1	0	0	1	2	A					
373	854027	5407/06	Kannivel	m	H	MN	A,S		L	G,D				NV	0	0	0	0	1	1	A					
374	853878	6049/06	Manoharan	m	H	MN	A,S		E	O,G,D				NV	0	0	0	0	1	1	A					

S. No	IP. No.	GE	Name	Sex	Presentation	Severity of bleed	Associated factors	Comorbidity	Timing of endoscopy	Endoscopic findings	Forrest Class	Sarin class	Varices-grade	V/NV	Rockall-AGE	Rockall-SHOCK	Rockall-Comorbidity	Endoscopic SRH	DIAGNOSIS	TOTAL Rockall score	Risk group	Blood transfusion	Endotherapy	Rebleed	Endotreapy for rebleed	Death
375	853892	6073/06	Sivaprakasam	m	H	MN	A		E	DU	1B			NV	1	1	0	2	1	5	B	B		Y	A	
376	854240	55/06	Santhi	f	H,M	MD		L	L	DU	2B			NV	0	2	2	2	1	7	C	B	A	Y	A	
377	854610	2106/06	Bharati	f	H	MD	S		E	PHTG				NV	0	2	0	0	1	3	B					
378	854390	6079/06	Ramesh	m	H	MD	S		E	O,G,D,DU	3			NV	0	2	0	0	1	3	B					
379	853109	6036/06	Indrani	f	H	MN			E	O,G,DU	3			NV	0	0	0	0	1	1	A					
380	855701	6198/06	Ramaiah	m	H	MN	S		L	H,O,G				NV	0	0	0	0	1	1	A					
381	855402	6171/06	Balu	m	H	MD	A,B,S	L	E	G,DU	2C			NV	0	2	2	0	1	5	B	B				
382	855688	6047/06	Alagu	m	H	MD	B,S		L	G,DU,GU	3			NV	0	1	0	0	1	2	A					
383	851421	6057/06	Gnanasekaran	m	H	MN	A,S	L	E	O,DU	2B			NV	0	1	2	0	1	5	B	B	A			
384	855752	6199/06	Krishnan	m	H	MD	S		E	O,DU,A	1B			NV	0	2	0	0	1	3	B	B	A			
385	856554	6277/06	Kuppammal	f	H,M	MD			E	O,DU	1B			NV	0	2	0	0	2	4	B	B	A			
386	856614	6291/06	Nisha	f	H	MN			L	O,G				NV	0	1	0	0	1	2	A					
387	857173	6352/06	Dhanona	f	H	MN			E	A				NV	0	1	0	0	1	2	A					
388	857415	6313/06	Subramani	m	H,M	MD	N		E	O				NV	0	1	0	0	1	2	A					
389	857847	6414/06	Annammal	f	H	MN	N		E	O				NV	1	1	0	0	1	3	B	B				
390	857948	6385/06	Podimal	m	H	MN	A		L	G				NV	0	1	0	0	1	2	A					
391	857860	6422/06	Jothi	f	H	MN			L	O				NV	0	2	0	2	1	5	B					
392	857555	4998/06	Kubendran	m	H,M	MD	B		E	DU	1B			NV	0	2	0	2	1	5	B		A	Y	A	
393	858400	6447/06	Dillibabu	m	H	MN	N,S		E	MS				NV	0	1	0	2	1	4	B					
394	858786	6493/06	Mohan	m	H	MD	A,S	L	L	G,D				NV	0	1	2	2	1	6	C	B		Y		
395	859660	6525/06	Rukmani	f	H	MN			L	G				NV	0	0	0	0	1	1	A					
396	858389	6585/06	Sonam	f	H	MD		L	L	G,D				NV	0	1	2	0	1	4	B					
397	859570	6574/06	Murugan	m	H	MD			L	G,D,DU	2C			NV	0	2	0	2	1	5	B	B				
398	860610	6628/06	Rajalaxmi	f	H,M	MN		O	L	G,D				NV	1	0	2	0	1	4	B					
399	860559	6639/06	Deenadayalan	m	H	MN			E	DU	1B			NV	1	1	0	0	1	3	B		A	Y	A	
400	861101	6684/06	Yesurathinam	m	H	MN	N		E	G				NV	0	0	0	0	1	1	A					
401	861239	6475/06	Gopi	m	H	MD	S		L	DU	1B			NV	0	2	0	2	1	5	B	B				
402	860988	1378/05	Kalpana	f	H,M	MD			E	A				NV	0	2	0	2	1	5	B	B				
403	860270	6775/06	Ponmudi	m	H	MN	A,S		E	G,DU	2C			NV	0	2	0	2	1	5	B	B				
404	862031	6776/06	Raja	m	H	MN	A,S	L	E	G,GU	1B			NV	0	1	2	0	1	4	B		A	Y	A	
405	862029	6741/06	Pari	m	H	MN			L	G,GU,DU	2B			NV	0	1	0	2	1	4	B		A			
406	861234	3560/03	Thayal nayaki	f	H,M	MD			E	O,G				NV	0	1	0	0	1	2	A	B				